8-19-2011 12:00 AM

The Use of Capacity as an Indicator of Automatic Processing: Is Smoking Automatic?

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A thesis submitted in partial fulfillment of the requirements for the degree in Doctor of Philosophy

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(Spine title: Smoking Behaviour and Capacity)

(Thesis format: Monograph)

by

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Graduate Program
in
Psychology

A thesis submitted in partial fulfilment
of the requirements for the degree of
Doctor of Philosophy

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entitled:

The Use Of Capacity As An Indicator Of Automatic Processing: Is Smoking Automatic?

is accepted in partial fulfillment of the
requirements for the degree of
Doctor of Philosophy

Date

Chair of the Thesis Examination Board

Abstract

It has been suggested that substance use transitions from a controlled to an automatic process (Tiffany, 1990). In particular, smoking has been found to appear automatic (Baxter & Hinson, 2001). Experienced smokers were able to attend to a reaction time task with minimal interference while smoking. Novice smokers' performances were impaired when smoking. These results were based on differences in mean reaction times using analysis of variance. Another analytic approach to test the hypothesis that smoking is an automatic process is through the use of capacity coefficients and ratios. These mathematical tools allow for the direct testing of mental processing. The goal of the current study was to use these capacity measures to investigate whether smoking taxed capacity, and whether results from this type of analysis differed from a traditional ANOVA. Also, capacity ratios were compared between smokers with different patterns of alcohol and cigarette co-use. There is a well established relationship between alcohol and cigarette use, and alcohol use itself may have an impact on smoking behaviour. Capacity ratios indicated that smoking does tax capacity, even in daily smokers. It was also found through the use of a pseudosmoking condition, in which the smoking behaviour had to be ceased partway, that the inhalation of smoke seems to require its own cognitive processing. When these results were compared to a traditional ANOVA, it was found that measures of capacity provided additional information. When smokers were grouped based on cigarette and alcohol co-use, results supported the hypothesis that smokers who frequently coupled cigarettes and alcohol were impaired in processing in comparison to less frequent couplers.

Keywords: smoking, automatic processing, capacity,

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Tobacco, alcohol, marijuana, cocaine, heroin, amphetamines, and hallucinogens are only a few of the psychoactive substances that people use in order to alter their experiences and consciousness (Jung, 2001). Reasons for the use of such substances are varied. People may use these substances to enhance positive feelings or to avoid negative feelings, because their peers use them or due to boredom. In addition to the reasons promoting the use of such substances, there are reasons that should dissuade people from using them. All substances have possible negative effects. These effects may be physiological, emotional, social, financial, or any combination of these. Yet, people continue to use substances despite the possible consequences. Some people continue to smoke tobacco after suffering heart attacks and strokes. Despite the risk of contracting HIV or hepatitis C, people share needles to inject themselves with drugs. Others go into debt to purchase substances. The fact that dire consequences do not dissuade people from using substances is puzzling.

One of the primary goals of substance use research is to understand the reasons why people use and abuse substances, despite the numerous negative effects that can occur because of substance use. A better understanding of these causes may lead to better treatment of substance use problems. Some of the factors involved in substance use that have been studied are the physiological effects that maintain substance use, the emotional factors involved in substance use, and how substance use interacts with other aspects in a person's life. However, despite all of the knowledge in the area of substance, there remain numerous unexplained aspects involved in substance use.

It is unlikely that one area of research will be able to comprehensively explain why people use psychoactive drugs. Substance use is multi-faceted, with many factors interacting with one another. While it is important to understand the immediate physiological effects of these drugs, these effects do not explain why someone is compelled to try the substance in the first place. Identifying risk factors, such as parental and peer drug use, does not explain why substance abuse may remain a problem when these influences are no longer present in an individual's life. While it is important to understand all of these contributing factors, it is clear that the search for reasons for the

initiation and continuance of substance use needs to be expanded. The role of cognitive factors in substance use has been a recent focus of study (McCusker, 2001).

Cognition is defined as the acquisition, storage, transformation and use of knowledge (Matlin, 1998). One way of conceptualizing cognition is the distinction between explicit and implicit. Explicit cognition involves the processes that we have awareness of, such as decision- making, purposively paying attention to a task, and manipulating information (e.g. adding or subtracting numbers). Arguably, most individuals have at least some explicit knowledge about various substances and their effects. People likely know that drinking alcohol may lead to intoxication. They may know that certain substances are illegal. They may use this knowledge in deciding whether to use a substance. Explicit cognition likely plays a role in the initial decision to try a substance, in that people weigh the reasons for and against its use.

The other type of cognition is implicit. Implicit cognitive processes have been defined as "introspectively unidentified (or inaccurately identified) traces of past experience that mediate feeling, thought, or action" (Greenwald & Banaji, 1995). Individuals acquire knowledge without realizing it, and this knowledge impacts their behaviours. It is possible that implicit cognitive processes are involved in substance use. Most people have explicit knowledge about why they should not use substances. If they were to weigh the reasons for using substances versus not using substances, it would seem that the scale should tip in favour against using these substances. However, something drives people to use substances despite what seems to be a clear inequality of positive and negative reasons. Implicit cognitive processes may be tipping the scale in favour of using these substances. While the person cannot state why they continue to use substances that harm them, something continues to drive the substance use. The recognition and identification of implicit cognitive processes may be important in the treatment of substance use problems. Making people aware of these implicit processes may allow them to counteract them, and tip the scales in favour of ceasing substance use.

One of the most common and difficult to treat addictive behaviours is the smoking of

tobacco. Despite the detrimental health consequence of smoking, 18% of Canadians smoke cigarettes (Health Canada, 2009). Quitting smoking is challenging, and even when quit attempts are successful, relapses are common (Balfour, 2004; Wetter et al., 1999). Cessation aids that target the physiological addiction to cigarettes, such as the nicotine patch, nicotine gum, and Champix, as well as behavioural treatments, have had only limited success (Hatsukami & Mooney, 1999). A combination of counselling and nicotine replacement therapy is more effective than counseling alone (Mojica et al., 2004), however quit rates remain relatively low. While there is a plethora of information available about the harmful health consequences attributable to smoking and the benefits of quitting, people continue to smoke. This indicates a disconnect between "knowing" and "doing" within smokers. While the explicit knowledge of the benefits of ceasing the behaviour are known, other factors continue to drive the smoking behaviour.

Cognitive processes that are outside of smokers' awareness may be involved in smoking, given that many experienced smokers report that they do not even think about smoking. Smokers report that they find themselves smoking without even remembering lighting up, and that they light up cigarettes without realizing that they already have a lit one in the ashtray (Hudmon et al., 2003). On a self-report measure of dependence, smokers positively endorse the item "My smoking is automatic- I don't even think about it" (Johnson et al., 2005). This lack of awareness is especially striking when considering the number of behavioural components that are involved in smoking.

A smoker must first obtain a cigarette from a package, ensuring that the cigarette is not broken in the process. The next step would be to place the cigarette in their mouth, which involves raising the cigarette to the mouth, slightly opening the mouth, placing the cigarette in the mouth, and then closing the mouth. Then the smoker must light the cigarette, which involves bringing an igniting source to the cigarette, draw in on the cigarette, and take the igniting source away from the mouth, making sure that they do not burn themselves in the process. The source of ignition may be a lighter or match. Each of these have their own behavioral components such as disengaging a child lock on a lighter, or pulling a match out of a matchbook.

Once the cigarette is lit, only then can the administration of nicotine begin, which entails inhaling smoke from the cigarette, drawing the smoke into the lungs, exhaling the smoke, and bringing the cigarette to and from the mouth, while shaking the burned tobacco off the cigarette at times that the smoker judges appropriate. On each inhale, the smoker must judge the optimal amount of smoke to inhale, hold it within the lungs for a certain period of time, and then exhale the smoke. The smoker must monitor the amount of the cigarette remaining to be smoked, so that the filter is not burned. The cigarette then must be discarded, which may involve extinguishing it and tossing it away.

Given the complexities of the behaviours needed to smoke a cigarette, it seems reasonable to assume that there must be some cognitive mechanisms underlying all of these behavioural components. Initially, either an explicit or implicit cognitive process needs to occur for the smoking behaviour to be initiated. Then, some degree of attention, another cognitive process, likely needs to be allotted to the pacing of moving the cigarette up to and away from the mouth, and also to ensure that one does not burn themselves. So while smoking is an overt observable behaviour, there are numerous cognitive processes underlying these behaviours.

In spite of the seeming complexity of smoking, experienced smokers perform a variety of other activities while smoking cigarettes. An experienced smoker may drive a car, read, or have a conversation, all while smoking. It appears that smoking is effortless. However, this level of ease is not present when a novice smoker first attempts these behaviours. The level of coordination needed to smoke is apparent when watching a novice smoker experiment with their first cigarette. This initial experimentation with smoking is usually accompanied by problems in lighting the cigarette, difficulties in pacing the inhalation and exhalation of smoke, or choking on the smoke.

Given that smoking arguably appears to be a cognitively consuming task, with intricate behavioural components, what enables an experienced smoker to smoke without any self-reported realization? How can an experienced smoker do other complicated tasks, such as drive a car, while smoking? These issues may be explained in the context of implicit

cognitive processes. Initially, the novice smoker may use explicit mechanisms to make the decision to have a cigarette and to deliberately attend to the components of the process. Over time, this may shift to an implicit process. The smoker no longer needs to actively decide to smoke or to pay attention to their smoking actions, and this frees the smoker to do other things while smoking. The smoking appears to occur "automatically".

Automatic and Controlled Processing

It has been argued that repeated substance use may transition from an initially controlled process to an automatic one (Tiffany, 1990). Broadly defined, controlled processes are those that require effort, attention, and awareness. Automatic processes are those that occur without awareness, attention or effort on the part of the actor. The initiation of substance use, such as smoking, is qualitatively different from use after many repetitions. This can be observed in the differences in smoking behaviour between a novice smoker and an experienced smoker. The speed, apparent ease, and need for attention of the smoker vary between the novice and the experienced smoker. This transition occurs after repeated practice. Tiffany argues that this difference in behaviour is explained by the shift from controlled to automatic processing.

Repetition of a cognitive or motor task under similar conditions results in the development of a skilled behaviour that is qualitatively different from when the behaviour was initially performed (Tiffany, 1990). Tiffany argues that this transition from controlled to automatic behaviour occurs in substance use. Behaviours, such as cigarette smoking, involve numerous components, such as taking the cigarette out of its package, placing it in the mouth, lighting the cigarette, and inhaling. All of these steps within the behaviour are repeatedly practiced. The processes are eventually stored in memory as action schemata (Schmidt, 1975; Shallice, 1972) or action plans (Allport, 1980; Newell, 1978). Automatic action schemas involve stimulus configurations, procedures, action sequences, alternative action sequences, and physiology (Tiffany, 1990).

The stimulus configurations may be external or internal events needed for the automatic process to be initiated (Tiffany, 1990). These may include environmental locations, time of day, or physical and mood states. Procedures include all of the actions that comprise the schemata. Action sequences coordinate all of the separate behaviours into one behaviour sequence. As mentioned above, smoking behaviour is comprised of various steps. The action sequence encompasses all of these behaviours. Alternative action sequences include contingency plans in the event that there are unexpected environmental conditions. Tiffany (1990) also outlines two physiological components of action schemata, support physiology for action components and physiological adjustment in anticipation of drug intake.

In order for these substance use action plans to develop, several conditions must be present. The first component is the availability of the drug. If obtaining the drug requires extensive planning, then this will most likely remain a controlled process. However, this does not prevent the administration of the drug from becoming automatic. Another component that affects the automatization of drug use behavior is environmental factors that may affect use. In the example of smoking, non-smoking legislation severely restricts the rights of smokers. Most public places prohibit smoking. This needs to be incorporated into the action plan, i.e., need to go outside to have a cigarette.

Lastly, Tiffany (1990) argues that characteristics of the drugs themselves may affect the development of action plans. Many drugs have inherent reinforcing properties. The administration of nicotine leads to the release of dopamine, norepinephrine and beta-endorphins. Tiffany argues that these positively reinforcing properties of certain drugs may promote more rapid development of coherent and integrated drug-use action schemata. He does acknowledge that this is purely speculative and that limited research has been conducted on the effect of reinforcing properties in the development of any automatic behaviour.

Tiffany (1990) illustrates the concept of the transition of controlled processing to automatic with the analogy of learning to drive a car. An inexperienced driver initially must devote conscious attention to all of the tasks involved in driving a car. They must

pay attention to which pedal to press, which way to adjust the indicator, how much they need to turn the steering wheel, and a number of other behaviours. With practice, the driver becomes accustomed to all of the steps required to drive the car. Eventually, the process becomes so automatic that the driver is able to direct his or her attention to other tasks such as changing radio stations or having a conversation, with little or no detriment to the driving itself. Of course, driving a car still entails some elements of attention. There is relative agreement that automatic vs. controlled processing is not an all-or-none phenomena (Moors & DeHouwer, 2006). Most processes involve a combination of both automatic and controlled processing, but distinctions may be made between the two.

It has been suggested that automatic processes are autonomous, unintentional, uncontrollable, purely stimulus driven and unconscious. Moors and DeHouwer (2006) have suggested that each feature of automaticity should be investigated individually. This approach would allow identification of those features that are present across all types of automatic processes. For example, autonomy has been suggested as the minimal criterion for all automatic processes (Bargh, 1992). Once an automatic process has been started, there is no need for conscious guidance or monitoring for it to be completed. However, there may be other features that are only present in certain types of automatic processes. Substance use may have unique components of automaticity, which are not present in other types of automatic processes. There may be differences in automatic features across different type of substances. Taking a features approach in the study of automatic processing allows us to be more precise in our definition, and empirical testing, of these concepts.

Empirical Evidence for the Controlled vs. Automatic Distinction

The historical roots of the empirical distinction between automatic and controlled processing come from cognitive psychology. Researchers initially focused on the apparent automaticity of cognitive processing. For example, Schneider and Shiffrin (1977; Shiffrin & Schneider, 1977) used an adapted Sternberg (1966) task to test whether repeated practice changes the processing of a visual stimulus. During the Sternberg task,

participants must decide whether a current visual target was included in an original memory set of items. For example, a participant may initially see the memory set "F T R K". They would then be shown a number of visual targets, and asked to indicate whether each target had been in the original memory set. Stimulus items that were included in the original sets were considered targets (e.g., the letter "F"), while items that were not in the original set were considered distracters (e.g. the letter "A"). After a number of trials with one memory set, participants would learn a new memory set of items and repeat the task. For example, the new set could be "L O Q A". Shiffrin and Schneider (1977; Schneider & Shiffrin, 1977) tested whether processing the stimulus item differed under two conditions; consistent and varied mapping.

Consistent mapping occurs when the target and distracter stimuli are consistent across the memory sets, e.g. the letter "F" would only be a target, and never a distracter. It was hypothesized that this type of mapping would eventually lead to "automatic" processing of the set. Initially, participants would need to compare the target stimulus to each item in the original memory set and decide whether it was a target or a distracter. If the initial set was "N R W F", and the stimulus was "F", the participant would need to recall all four items in the set to determine whether an "F" had been present. The next memory set may be "Q F H J" where F is again a target. Over conditions, determining that "F" is a target would arguably become quicker, easier, and require less attention. When one saw the letter "F", they would automatically know that this was a target. The recall of all original items is no longer required since it is now automatic that "F" is a target.

Varied mapping occurs when stimuli can both be targets and distracters across conditions. For example, in the first condition the memory set may be "K L U F". In that case, "F" is a target. In the next condition, the set could be "P Q W X", so that "F" is now a distracter. The participant must continually compare the target to the original set. Under these conditions, the processing of the target is deemed to be slow, effortful and requires attention. Practice should not have much effect on processing speed, as the status of each stimulus item continuously changes.

In a series of experiments using the adapted Sternberg task, Schneider and Shiffrin (1977; Shiffrin & Schneider, 1977) showed there were distinct differences in performance between the consistent and varied mapping conditions. Processing was much quicker in the consistent mapping condition than in the varied mapping condition. Participants were faster to respond in the consistent mapping condition whether the stimulus item was a distracter or a target. Shiffrin and Schneider argued that the cognitive process had become more automatic in the consistent mapping condition. Processing remained controlled in the varied mapping condition. This provided evidence that one of the features of automatic processing is enhanced speed.

In further experiments using the Sternberg task, Schneider and Schiffrin (1977; Shiffrin & Schneider, 1977) manipulated the number of items, up to a maximum of six, in the original set. They found that the number of items in the original item set had no effect on performance in the consistent mapping conditions. Performance was the same, regardless if the original memory set had 4 items or 6 items. The number of items in the memory set in the varied mapping condition impacted performance dramatically. In the varied mapping condition there were increases in reaction time as the number of items in the memory set increased. The lack of variation in performance despite increases in the number of items in the memory set in the consistent mapping conditions was argued to be evidence of the efficiency of automatic processing. An automatic process is not impeded by changes in the load of items to process.

The transition from controlled to automatic processing is argued to occur as a function of practice. Schneider (1985) outlined four phases that occur when a process shifts from controlled to automatic processing. Controlled processing is the first phase. Performance is slow, serial and effortful. It is also greatly impacted by increases in memory or processing loading. The second phase occurs after consistent practice. The amount of practice required for this shift may vary across tasks. A mix of both controlled and automatic processes characterizes this stage. Phase three is classified as automatic processing with controlled-processing assist. Although individuals may occasionally attend to the task they are doing, the need for full attention is greatly decreased. The final

phase in the transition is full automatic processing.

Schneider (1985) illustrated this model using a category-search experiment. The participant was presented with a short list of categories, such as Animal and Tree. They were then shown words that may or may not be members of the previously shown categories. The task involved the participant responding with a motor response, indicating whether the target words are members of one of the categories. In the first phase, controlled processing, the participant has many tasks to complete. They first have to remember the categories initially presented to them (e.g., Tree and Animal). This is considered the semantic vector within the model. They are shown target words (e.g. Cat and Car), which is the visual vector. They must then make a response, which is the motor vector.

The semantic vector is preloaded with the names of the categories. The number of categories presented to the participant will affect the speed of the process. This is considered the effect of memory loading. When the target words are displayed, this activates the visual vector. The number of words that are presented will affect the speed of the process. A greater number of words presented will slow the processing speed. Once the words are presented, this will activate the memory unit for the categories shown previously. The information presented visually is then processed through the semantic criteria (i.e. is one of these words part of a previously seen category?). Once a decision has been made, a motor response must be made. This activates the motor vector. There are numerous steps within the model. When this task is first presented, processing will be very effortful, slow, and controlled.

The second phase in Schneider's model is defined as the co-occurrence of controlled and automatic processing. Once the target word is shown, a motor response will be associatively evoked. There is still the need for controlled processing of the early stages of the task (seeing the categories, remembering them, seeing the target word, making a decision), but the response to the task becomes more automatic (learning that the index finger is "yes" and middle finger is "no"). During the third phase, there is a shift to

automatic processing with controlled-processing assist. The subject attends to the task in general, rather than each step separately. The participant may generally do the task automatically for the majority of words, but switch to controlled processing for a subset of less common words. For example, the response may be automatically "yes" if the category is Animals and the target word is Cat. However, there may be a delay in response if the word is something novel, such as Marsupial. The final phase is automatic processing. Schneider notes that there is no clear transition between phase 3 and 4.

Automaticity occurs in a series of phases. It is only through repetition and practice that a task becomes automatic. A complex series of decisions and behaviours occur with greater ease and effortlessness over time. Does this transition occur with substance use? Is the development of substance use even comparable to these traditional cognitive tasks?

Testing Whether Substance Use is an Automatic Process

It has been suggested that substance use may be conceptualized as an automatic process. Characteristics of automatic processing, such as speed, autonomy, a lack of control, effortlessness and a lack of conscious awareness have been described as characteristics of substance use (Tiffany, 1990). There does appear to be a qualitative difference in the smoking of novice and experienced smokers. Experienced smokers themselves identify their smoking as automatic (Johnson et al., 2005). However, many issues need to be considered before deciding whether smoking, or any other substance use, is an automatic process.

The first issue is defining automaticity. As previously discussed, there is no consistent definition for automaticity (Moors & DeHouwer, 2006). There is a need for both conceptual and empirical precision in the definition of automaticity. Developing a definition of automaticity may involve identifying features that are characteristic of automatic processes. Much of what we do throughout the day appears to occur automatically, without our conscious awareness (Bargh & Chartran, 1999). However, it is not enough to describe something as automatic simply because it appears to be so. The

way in which automaticity is defined will have an impact on the experimental design when testing for automaticity.

In addition to the definition of automaticity, testing substance use experimentally has its own set of challenges. Many substance use behaviours are difficult to bring into a laboratory setting. Situational factors that impact substance use, such as peer groups or the environment, are difficult to replicate in a laboratory. There are ethical constraints when conducting laboratory research. Researchers must ensure participants' safety, and it is complicated to do so if administrating a substance that produces intoxicating or deleterious effects. Many substances are illegal, which may be a challenge in both acquiring ethical approval to study and provide challenges in obtaining the substance itself. Potential participants may also be hesitant in participating in such research. Smoking is likely the easiest substance use behaviour to study in a laboratory setting given that smoking is legal, cigarettes are easily accessible, and arguably have minimal, if any, acute debilitating effects.

There are some potential problematic issues in observing smoking in a laboratory setting. Most of the external factors that may influence smoking, such as seeing others smoke, eating, and environmental factors (Shiffman et al., 2002), are removed. One of the more salient co-activities that may have an effect on smoking is the use of alcohol. The relationship between cigarette and alcohol use among regular smokers is robust. Approximately 80-90% of smokers drink regularly, and smokers are heavier drinkers than non-smokers (Strine et al., 2005). There is evidence that there is a pharmacological interaction between alcohol and cigarette consumption. In laboratory studies, drinking increases the rate and amount of smoking (Mintz, Boyd, Rose, Charuvastra & Jarvik, 1985), and transdermal nicotine administration increased alcohol consumption in men who smoked 1 to 10 cigarettes per day (Acheson, Mahler, Chi, & de Wit, 2006).

There is also a relationship between light smoking and alcohol use. Smokers who smoke 2-8 cigarettes a day smoked a greater proportion of cigarettes in the presence of alcohol than those who smoked more than 10 cigarettes a day (Krukowski, Solomon, & Naud,

2005). Ninety-four percent of those who smoke fewer than 5 cigarettes per day at least 5 days a week (referred to as "chippers"), reported smoking while drinking alcohol (Shiffman, Kassel, Paty, Gnys, & Zettler-Segal, 1994a; Shiffman, Paty, Kassel, Gnys, & Zettler-Segal, 1994b). Situations in which alcohol is consumed may be particularly prone to induce smoking in individuals who do not smoke on a regular basis. Smokers who only or primarily smoke when using alcohol (high co-users) may be different from smokers who use cigarettes in a broad range of situations (low co-users), in terms of dependence levels, frequency and quantity of smoking.

In addition to these differences between low and high co-users of alcohol and cigarettes, alcohol may have an impact on whether smoking becomes automatic. There is a long history of research examining the effects of alcohol's influence on learning. Alcohol intoxication has been found to impact learning, particularly when information is shifted from short- to long-term storage (Weingartner & Faillace, 1971a, 1971b). This may impact the storage of all of the components of smoking behaviour. As described earlier, smoking does involve numerous behavioural components. While alcohol may not impact the initial learning of smoking behaviour, it may impact its transition to an automatic behaviour.

Using multinomial processing tree models, Chechile (2010) investigated the impact of alcohol use on the storage and retrieval of information following acute alcohol ingestion. He found that alcohol use impairs the storage of new information, and suggested that alcohol impairs the initial quality of memory encoding. However, there were minimal differences in retrieval between alcohol and placebo use. Chechile, however, cautioned that these results should not be interpreted as evidence that alcohol never impacts retrieval, as the retrieval task was done shortly after learning the new information. These results may have differed if the time between learning and retrieval was increased, as is the case with smoking and alcohol. It is likely that there are relatively long lags between smoking opportunities.

Another consideration of the relationship between alcohol and cigarette use is state-dependent learning. It has been found that information that is learned when intoxicated is more easily retrieved under intoxication in comparison to sober conditions (Goodwin, Powell, Bremer, Hoine, & Stern, 1969.; Weingartner, Adefris, Eich, & Murphy, 1976). If smoking behaviour is learned when using alcohol, then it may be more difficult to smoke in non-drinking circumstances.

Dual Task Paradigms

One of the primary methods of studying whether a behaviour or process has become automatic is the dual task paradigm. This technique involves determining if there are limitations in performing two tasks at once. Cognitive psychologists argue that humans are limited in their ability to do multiple tasks at once well (Band, Jolicoeur, Akyürek, & Memelink, 2006). This difficulty may be a result of an inability to divide attention, a lack of capacity, or a deficiency of cognitive resources. Typically, performance on two concurrent tasks is decreased in comparison to attending to only one task at a time. However, the dual task paradigm has been used to investigate whether there are conditions under which this decrease in performance does not occur. Typically, performance on a novel primary task is measured on its own and then with a concurrent secondary task. Performance is usually impaired on the primary task, with the introduction of the secondary task. The primary task is then extensively practiced, and the secondary task is reintroduced. Following practice, performance on the primary task is no longer affected by the secondary task. It is hypothesized that the primary task has become automatic and attention can be diverted to the secondary task.

While dual task paradigms are widely used, there are no standard formats for their use. The natures of the tasks vary substantially from study to study. Some examples of dual task paradigms include pressing a button when a target symbol is presented while repeating digits that have been presented verbally (Karatekin, Couperus, & Marcus, 2004), tracking a light with a stylus while pressing a button every 5 seconds (Brown & Bennett, 2002), or determining if presented numbers are odd or even while determining

whether combinations of letters were words or non-words (Waters & Green, 2003). The dependent variable is most often reaction time, but accuracy of responses may also be measured. Performance is usually measured on both tasks, which is usually poorer than when doing each task on its own. However, it has been found that after practice on one of the tasks, dual task interference is reduced (Eysneck & Keane, 2010). The decreased dual task interference is posited to occur because the well-practiced task becomes automatic and thus taxes fewer cognitive resources.

The amount of practice required for a process to be deemed automatic in an experiment varies from study to study. The amount of practice can range from a number of hours (Wu, Kansaku & Hallett, 2004), to days (Kubler, Dixon & Garavan, 2006), to weeks (Bebko et al., 2003). The criteria for deeming a behaviour as automatic again varies from study to study, and is largely dependent on the type of task. The criteria may be engaging in the behaviour with total accuracy or increasing speed by a certain percentage. Other studies merely consider the lack of dual task interference as evidence of automaticity. These inconsistencies across studies again stress the need for a more refined definition of automaticity, which may be accomplished through the use of a feature based approach.

Another difference between traditional dual task studies and any study of whether smoking is an automatic process is controlling the amount of experience people have with the smoking behaviour. In most dual task research, the studied behaviours are initially novel, such as juggling (Bebko et al., 2003), and the researcher can control the participants' amount of practice and level of performance. Smoking, however, is not a behaviour that can be practiced in the lab. Each person has his or her own unique history with smoking. Some individuals may have had their first cigarette years ago, while others may have only started smoking within the last few months. Some smokers are daily smokers, while others may only smoke on weekends. There are many possible differences in smoking history and current smoking behavior. Given this variability in smoking, it will be impossible to directly study the development of smoking behaviour. We can only rely on self-reports of smoking to ascertain experience. However, by using the dual task paradigm, it is possible to determine whether the current experience of

smoking interferes with some other cognitive task.

Baxter and Hinson (2001) used the dual task paradigm in an investigation of smoking as an automatic process. Participants were trained to recognize a target tone. They then listened for the target tone under four separate conditions; no smoking, smoking, pseudosmoking, and holding a cigarette. The participants were classified as experienced smokers (having smoked more than 800 cigarettes in their lifetime) or novice smokers (having smoked less than 100 cigarettes in their lifetime). The pseudosmoking condition involved performing all smoking behaviours except for inhaling the smoke of the cigarette. The premise behind this condition was that it would be difficult for experienced smokers to break the automatic action schema without diverting their attention to the smoking behaviour. Similar reasoning was held for the holding condition. As expected, experienced smokers' performance on the tone recognition task was similar in the smoking and no smoking condition. Performance was significantly impaired during the pseudosmoking condition. Smokers had to divert their attention from the tone recognition task to halt the smoking action sequence. In contrast, novice smokers' performance was equally impaired in both the smoking and pseudosmoking conditions.

As with almost all dual task research, Baxter and Hinson (2001) analyzed mean reaction times on the tone recognition task. Similar reaction times in the no smoking and smoking conditions for experienced smokers were interpreted as evidence for automaticity. Reaction times were greater in the pseudosmoking condition. Although significant differences were found in response times, it is not clear why these differences occurred. Tone recognition is not the result of a single cognitive process. There are numerous possible explanations for the differences in performance between conditions. Traditional statistical methods, such as Analysis of Variance (ANOVA), are useful for detecting differences between experimental conditions, but they are not designed to assess and compare the various contributions of underlying cognitive processes (Batchelder, Chosak-Reiter, Shankle, & Dick, 1997).

To gain a better understanding of any behaviour, one must go beyond the observation of the behaviour itself. Luce (1995, 1999) argues that the main difference between various models of behaviour is whether the "black box" remains closed or is opened. Behavioural observation is looking at the box closed – merely observing the inputs and outputs. There is no internal structure specified within the model. Luce argues that most psychological theories are of this nature; "they attempt to characterize aspects and patterns of behaviour without asking about the underlying, internal mechanisms that give rise to the behaviour" (Luce 1995, p. 3).

Information Processing Models

Information-processing models strive to open the black box of behaviour. These models attempt to describe a structure or architecture that information must pass through when received. This information then results in a response set, resulting then in some sort of feedback. Luce (1999) states that information processing models may be thought of as trying to describe analytically a flow diagram of mental activity based on certain elementary processing stages. The advantage of information-processing models over behavioural models is they provide natural accounts of temporal aspects in decision-making. It is possible to identify where errors occurred in the formation of an inaccurate response. Using input-output models, when an inaccurate response is made, one has no way to identify why the error has been made. There are numerous possibilities, such as problems with memory, retrieval, or making a response. However, it is impossible to know when only the response is examined. Using an information processing model approach, it is possible to identify the source of the error.

Townsend & Wenger (2004a) argue that much of the evidence for psychological theory is circumstantial and can only be said to be consistent with an assumption, but most of the time does not come from a direct test of that assumption. In their study of smoking as an automatic process, Baxter and Hinson's (2001) results appear consistent with the assumption that smoking in experienced smokers is an automatic process. Since performance was equal in both the smoking and non-smoking conditions in experienced smokers, this was taken as evidence for smoking as an automatic process. However,

there is no direct evidence that smoking is, in fact, an automatic process. All that is certain is that the output (reaction time) is the same for both conditions.

Information processing models, according to Townsend and Wenger (2004a), have four components; architecture, stopping rules, independence and capacity. Architecture may be serial or parallel. Serial processing occurs when each element is processed one at a time, each element being completed before the next is begun. Parallel processing occurs when processing begins on all elements simultaneously and continues until each element is completed (Townsend & Ashby, 1983). In addition to the distinction between serial and parallel models, there is also the consideration of a special kind of parallel processing called coactive processing models (Townsend & Nozawa, 1995). In separate parallel processing models, each process goes through its own channel. In coactivation models, processes are consolidated into a single channel.

Stopping rules refer to whether cognitive processing is self-terminating or exhaustive. A self-terminating process occurs when the mechanism responsible for the processing is able to stop when the desired element is found among all stimulus elements (Townsend & Ashby, 1983). Exhaustive processing occurs when all the elements are processed prior to cessation. Self-terminating processing is best illustrated by a target element being one of the first items in a set of elements. If the target element was F, and the set of elements was T F R Q J, processing would stop at F and there would be no processing of R Q J. Evidence for self-terminating processing is reaction times. Reaction times for the set T F R Q J would be shorter than for the set T R F Q J. Exhaustive processing can occur regardless if the target is present or absent. It could occur if the target set were J U R A H (target absent) or if the target set were T F R Q J (target present). Even though the target is found early in the target set, all remaining letters are processed. Again, evidence for exhaustive processing is the reaction times. Both sets would be processed in the same amount of time despite the fact that one set contains the target and the other does not. If processing were self-terminating, reaction times for the target present set would be quicker than for the target absent set.

Independence refers to the relationships among the rates of processing for each of the inputs (Townsend & Wenger, 2004a). If the information processing is independent, the processing of element A does not affect the processing of element B. No matter what occurs in the processing of element A, processing of element B will remain unchanged. A violation of independence occurs when the processing of a preceding target somehow affects the processing of the following target.

Capacity refers to the speed and accuracy of a processing system when its load is varied (Townsend & Ashby, 1983). There are three different possibilities for capacity. The first is unlimited capacity. If the processing system has unlimited capacity, increases in task load do not result in a decrease in performance. For example, if an increase in the number of targets in a memory set did not produce a change in reaction time, then capacity would be considered unlimited. In contrast, limited capacity is when processing load affects performance. Increases in workload push at processing limits. Finally, there is super capacity. This refers to an increase in performance ability when there is an increase in load.

The unlimited capacity, independent, parallel (UCIP) model (Townsend & Wenger, 2004) may be used in order to conceptualize how "automatic" processing may look from an information processing modeling perspective. If a process is automatic, it is reasonable to assume that capacity is unlimited. The processing requirement of one task does not impact the processing of another task. Similarly, automatic processing should be independent, in that the processing of one task does not affect the processing of another element. That automatic processing may require a parallel system is a reasonable assumption. If a process is automatic, its components would not need to be processed one at a time. However, it has also been suggested that automatic processing may be coactive (Townsend, Fific, & Neufeld, 2007) depending on the type of processing. The use of information processing models could be used in order to move towards a more features based approach of automatic processing.

Response Times in the Study of Information Processing Models

Response time (RT) research has a long history in psychology. RT research has served as a tool to study various cognitive processes and to more fully understand information processing models. By studying the length of time a particular task takes, and comparing it to the time other tasks take, one can make inferences about the processes involved. The first publication using response times was by Donders (1868/1969). He proposed the "method of subtraction". He argued that by subtracting the response time of a simple task from the response time of a more complex task, one could estimate that amount of time it took for the more difficult task. For example, a participant is shown a target element (e.g., the letter A). In the first recognition task the participant is shown two elements (F A). In a second task the participant is shown five elements (G J X F A). The difference in response times would represent the time that it took to process the three additional elements. This argument rests on the assumption that cognitive processes occur in a serial manner. In relation to the method of subtraction is the concept of pure insertion (Donders, 1868/1969). The underlying assumption of pure insertion is that adding a component to a task, does not change the operation of other tasks. The investigation of pure insertion has largely been unpopular, as it is practically impossible to test at the RT mean level (Townsend & Ashby, 1983).

Response times continue to be a useful tool in the measurement of cognitive processes. The evolution of mathematical modeling has allowed researchers to use RTs without the constraints of the assumption of seriality, as is the case with the method of subtraction. Some of the mathematical tools that may be used in the study of information processing models are the probability density function, the cumulative probability distribution function, the survivor function and the hazard function. There are an infinite number of probability laws and functions, including the normal and exponential (Townsend, et al., 2007). The probability density function [f (t)] indicates how completion probabilities change over time. Probability density functions are illustrated as a line curve, with reaction time on the X-axis and the function on the Y-axis, as illustrated in Figure 1. The cumulative probability distribution function [F(t)] represents the probability of an event

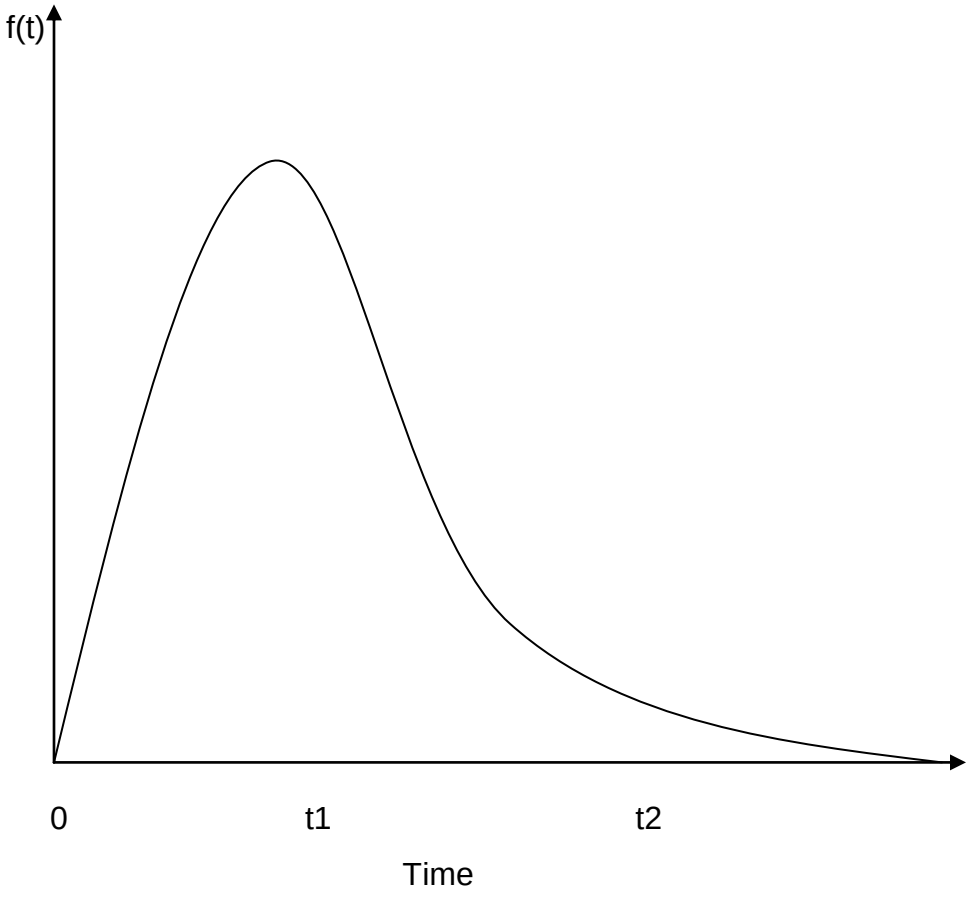

Figure 1. Example of a Probability Density Function (from Townsend & Ashby, 1983)

occurrence at or before reaction time t. The cumulative probability distribution function will always be either increasing or flat, its smallest value 0 and its largest value will be 1 (Wenger & Townsend, 2000). An example can be seen in Figure 2.

Another function of interest is the survivor function [S(t)]. This function indicates the probability that completion has not yet occurred. The survivor function is complementary to the cumulative probability distribution, and as such is calculated as 1 – F(t). Another function that is widely used in information processing modeling is the hazard function [h(t)]. The hazard function is a ratio of the density function over the survivor function,

$$\frac{f(t)}{S(t)}.$$

It indicates the probability of item completion immediately, given that it has not yet been completed. The hazard function comes from the concept of power in physics, and is conceptualized as the amount of power a system must use to transition a process from incompletion to completion (Wenger & Townsend, 2000). Neufeld, Townsend and Jette (2007) describe the hazard function as "a transitory function of time and therefore difficult to estimate empirically" (pg. 212). Therefore, when using the hazard function in order to calculate capacity measures, it is best to use the integrated hazard function H(t), which is a more reliable estimate of the amount of "energy expenditure" that takes place in cognitive processing. The formula to calculate the integrated hazard function is -ln[S(t)] where ln(x) stands for the natural logarithm of x. Other methods of calculating the integrated hazard function do exist, such as the Nelson-Aalen estimator (Aaalen, Borgan, & Gjessing, 2008), however -ln[S(t)] is most commonly used.

The integrated hazard function can be used to calculate both capacity coefficients and capacity ratios (CR). CR is used to compare performances between two conditions to investigate whether one condition is more capacity taxing than the other. The ratio

$$\frac{\mathrm{H(t)}condition\ 1}{\mathrm{H(t)}\ condition\ 2}$$

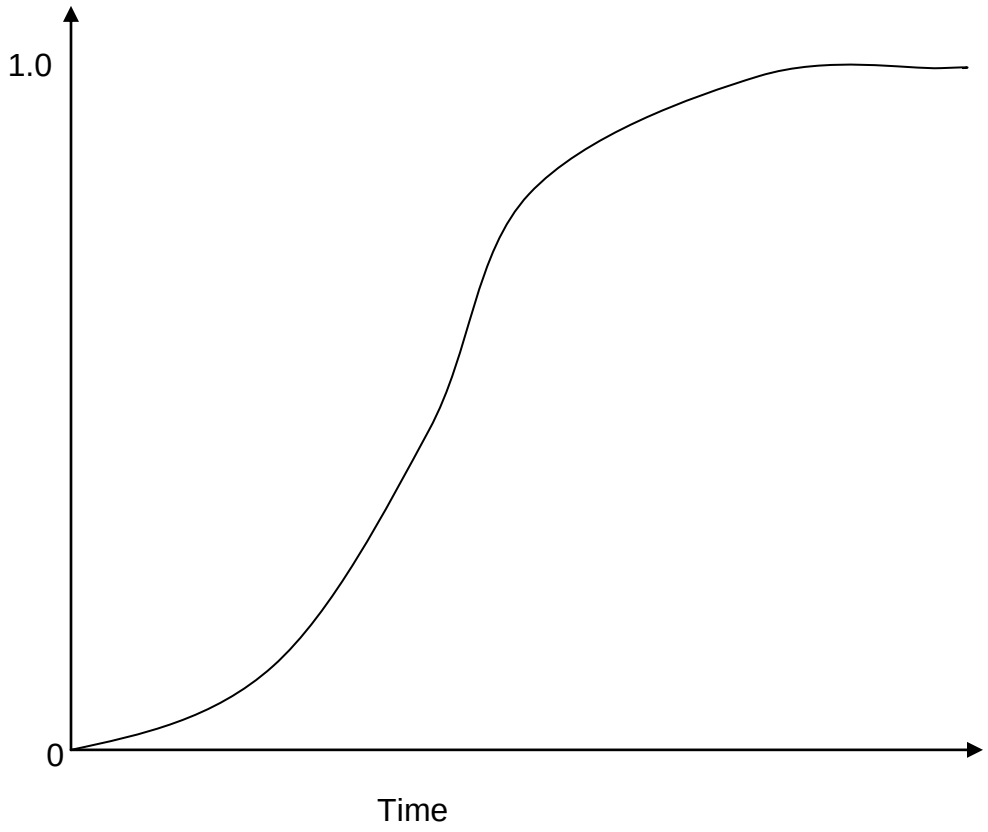

Figure 2. Example of a Cumulative Probability Distribution Function

would allow for comparison of processing between the two conditions. If the ratio equals 1 then that would indicate that cognitive processing in each of the conditions was equal, and the manipulation in condition 1 did not tax capacity in relation to the processing capacity of condition 2. If the ratio is less than 1 that would indicate that the manipulation in condition 1 taxed capacity more in relation to condition 2. Finally, if the ratio was greater than 1 that would indicate that condition 1 taxed capacity less than condition 2.

The use of CR can be demonstrated using a paradigm employed by Jette (1997). Participants were presented with six lamps, two placed centrally, two on the right periphery and two on the left periphery. They were instructed to indicate which lamp lit up on a six button panel. The response task was completed amidst one second bursts of white noise. The noise levels were of three intensities. The hypothesis was that performances would be improved in the more intense noise condition, as processing capacity was expected to increase with noise levels. (Neufeld et al., 2007). When comparing performance across noise intensity conditions, CR was calculated by

$$\frac{\mathrm{H(t)}lower\ noise\ \ level}{\mathrm{H(t)}higher\ noise\ level}$$

The capacity ratios were less than 1, indicating that processing capacity was more greatly taxed in the lower noise condition than in the higher noise condition. Performance was impaired in the low noise condition in comparison to the high noise level condition, which was consistent with a proposed mechanism of selectively improved performance under higher noise conditions.

Another method to use the integrated hazard function is the OR capacity coefficient, $C_o(t)$. While CR can measure capacity demands across conditions generally, $C_o(t)$ also takes into consideration specific increments in task load. The capacity coefficient is the ratio of the integrated hazard function when two features are present as the numerator, and the sum of the two integrated hazard functions for the conditions in which each feature is presented individually. If the capacity ratios equals 1, capacity is unlimited. If it is less than 1, this indicates limited capacity, and if it is greater than 1, it indicates super

capacity. The equation is (c = feature present in channel, 0 = feature absent in channel):

$$C_o(t) = \frac{H\,c,c(t)}{(H\,c,0\,(t) + H\,0,c(t))}$$

A use for this type of capacity coefficient is seen in a study of the processing of faces (Wenger & Townsend, 2001). Faces are made up of a number of features, including eyes, nose and mouth. It has been argued that faces are processed as a whole, rather than as sets of multiple features. Wenger and Townsend used the capacity coefficient to empirically test whether this was indeed the case. Participants were presented with faces with mouth and eyes simultaneously present, only the eyes present, only the mouth present, or no features present. Respondents were asked to respond affirmatively if either the eyes or mouth were present in all of the presentations. The capacity coefficients were less than 1 which indicated capacity was limited when both features were present in comparison to when only one feature was presented at a time. This was a surprising result and contradicted the expectation that faces were processed as a whole. Having multiple features taxed capacity in comparison to only having to attend to one feature at a time.

In the experimental design used by Wenger and Townsend (2000), participants were instructed to make a response if at least one of the features was present. This is an example of an OR design. The participant makes a positive response whether the target is in either of the channels. Theoretically this process is self-terminating as the participant may make a response if the target is in the first channel, obviating the need to process the second channel. Another experimental design is the AND design. In this type of design, respondents are instructed to respond only if a target is in both channels. In this design, exhaustive processing must be used since participants need to process both targets. The capacity coefficient for the AND design is

$$C_a(t) = \frac{(K\,c,0\,(t) + K\,0,c(t))}{K\,c,c(t)}$$

The capacity coefficient for the AND condition incorporates K(t), which Townsend and Wenger (2004b) argue is analogous to the integrated hazard function, H (t). While H(t) is the conditional likelihood that an event will happen momentarily given that is has not yet happened, K (t) is the conditional likelihood that an event occurred just before t, given that it has happened by time t. The calculation is

$$K(t) = \ln[F(t)] \leq 0$$

The calculation of H(t) relies on the survivor function, whereas the calculation of K(t) relies on the density function integral. According to Townsend & Wenger (2004b) the "inversion" in the AND capacity coefficient makes the interpretation of the coefficient identical to the interpretation of the $C_o(t)$ coefficient. A $C_a(t)$ value of greater than 1 indicates super capacity (an increase in load actually increases performance), a value of 1 indicates unlimited capacity (an increase in load does not affect performance), and a value less than 1 indicates limited capacity (an increase in load decreases performance. Townsend and Wenger also state that it is unclear whether K(t) will gain widespread popularity in mathematical modeling. The use of the AND coefficient is currently novel and its use in the current study is largely exploratory.

Smoking as an Automatic Process and Capacity

It is hypothesized that smoking does become an automatic process. While smoking contains a number of behavioral components that require attention, experienced smokers eventually are able to smoke while doing a secondary task. This suggests that with practice, the attentional need required for smoking decreases as the smoker gains more experience with smoking. Smokers' own reports of smoking being automatic (Johnson et al., 2005) also suggests that the need for attention decreases with smoking experience. Baxter and Hinson's (2001) results support the notion that smoking does become an automatic process.

As previously discussed, it has been suggested that the study of automaticity should be a feature-based approach (Moors & DeHower, 2006). Capacity can be conceptualized as a feature of automaticity. As found by Shiffrin and Schneider (1977), the number of items

in a memory set did not affect response time performance in the consistent mapping condition. The number of items included did not appear to tax capacity, although this is inferred based on mean reaction times and not an analysis of capacity coefficients.

While smoking may not resemble traditional cognitive tasks used in dual task research, the act of smoking and the cognitive processes that underlie it, should not tax the processing of a secondary task if smoking is automatic. Returning to the concept of pure insertion (Donders 1868/1969), the addition of smoking should not impact performance on a secondary task. The task should be completed identically, whether smoking is occurring or not. While mean reaction times cannot address pure insertion, the use of capacity ratios can provide as with evidence whether smoking is affecting the processing of the secondary task. In contrast, if the smoking act is guided by controlled processing it would be expected to tax capacity. The act of smoking for a novice smoker should be a relatively controlled task and should tax the ability to process a secondary task.

The purpose of the present analysis is to utilize capacity ratios (CR) to determine whether smoking does tax capacity. Capacity coefficients will also be calculated in order to determine the capacity demands of different types of stimulus presentations. Capacity ratios will be calculated to test whether smoking or pseudosmoking taxes capacity in comparison to no smoking. It is expected that for experienced smokers, the CR will be close to 1 when comparing performance during smoking and non-smoking conditions. This will show that the addition of smoking does not tax capacity. However, during the pseudosmoking condition, the CR should be less than 1, indicating that pseudosmoking does tax capacity in experienced smokers. For novice smokers, the CR for both the smoking and pseudosmoking conditions should be less than one when compared to the non-smoking condition since both of these behavior should be under controlled processing and thus should tax capacity.

The data will then be re-analyzed using an analysis of variance (ANOVA). The inclusion of this analysis is meant to serve as a comparison between the traditional ANOVA approach to evaluating differences in reaction times and the information processing

model approach. It is the goal of this comparison to evaluate whether the overall results are comparable, and whether the capacity ratios and coefficients do serve as a more fine-grain approach to evaluating reaction time data. Lastly, capacity ratios will be compared between smokers who primarily use cigarettes when using alcohol (high co-users) and those who do not (low co-users). As discussed in the introduction, the pairing of cigarettes and alcohol on a consistent basis may have an impact on the development of the automaticity of smoking behaviour.

Calculating Capacity Coefficients and Ratios

The following section is a detailed description of the procedures used to calculate capacity ratios and capacity coefficients. While it has been argued that capacity coefficients and ratios can be extremely useful in psychological research, their mathematical sophistication may be intimidating to the novice user.

Capacity coefficients and ratios may be used in various experimental designs, however, they will be illustrated in the context of the procedures used in the current series of studies. The task used in the current study is a modified version of one used by Egeth and Dagenbach (1991). There were two stimulus positions, one located on the left side of the computer screen (X_) and one on the right (_X). Participants were instructed to respond affirmatively if they saw the target X, and to make an alternate response if the target was not present. The non-target was O. Each of the two target positions may have included an X, O, or was left blank. The various combinations of stimuli can be seen in Figure 3.

Once reactions times were collected and incorrect responses were discarded, a number of issues needed to be addressed prior to proceeding with analysis. One of the criticisms of traditional statistical analysis is the treatment of outliers. Often the issue is ignored, or certain percentages on both tails of the distribution are discarded. However, it is often the outliers that are of most interest in regards to reaction times. In addition, reaction times often lack a normal distribution, but are positively skewed. Capacity coefficients are distribution general, so the analysis is not impacted by the shape of the distribution. However, there will be responses that are likely anticipatory or a result of dissattention (Neufeld, et al., 2007). For the current study, reaction times less than 100 ms and greater than 3000ms were discarded (personal communication, Neufeld). Wenger and Townsend suggest that no greater than 10% of data points should be discarded from the analysis (2000).

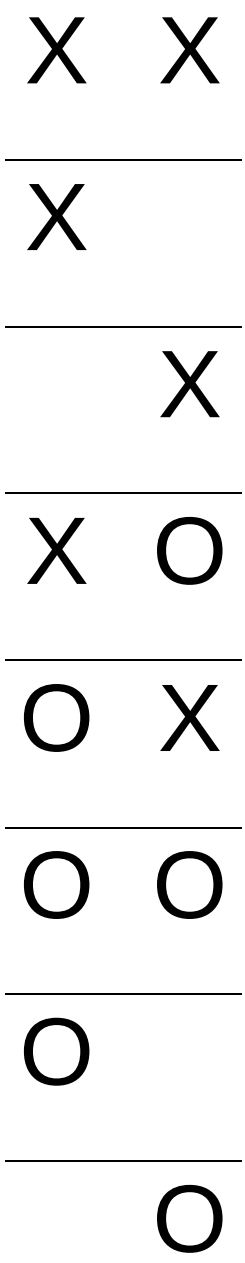

Figure 3. Stimulus Configurations

Once outliers are discarded, a certain amount of time needs to be subtracted to account for base processes. The base processes are those processes that are involved in making a response, but not part of the cognitive processes. These are mainly the rudimentary process involved in responding, such as the physical movements of pressing a response key (Carter, Neufeld & Benn, 1998). For the current study 160 ms were subtracted to account for base processes (Townsend, 1984; Woodworth, Schlossberg, Kling, & Riggs, 1971).

Once the data were prepared, reaction times for each variable were placed in bins. These bins are collections of reaction times. The bin values were adapted from Neufeld, et al. (2007) which used a boundary of 600 ms for the initial bin, and then increased by 200 ms for each subsequent bin. This binning procedure was based on the precedent of Hockey (1970). The initial bin in the current study was set at 0-400 ms. This decrease in bin size was used as the current study involved only two possible responses (target present – yes or no), whereas there were six possible responses in the previous study. It was hypothesized that given the decreased number of responses, processing time may be decreased and be more accurately captured in a smaller initial bin. The remaining bins were consistent with Neufeld et al.'s methodology and increased by 200 ms increments; 0-400 milliseconds, 400-600 milliseconds, 600-800 milliseconds, 800-1000 milliseconds.

When amalgamating data across participants, it is important to ensure that data within each group are homogenous. There are numerous sources of variance within data. These can be due to the process model, individual differences, base processes, and within-subject residual (Carter, Neufeld & Benn, 1998). For the current analysis, the process model variance would be the variance in capacity due to the experimental manipulations. In order to assess homogeneity, first a two factor analysis of variance is conducted. The two factors are the bins and smoking groups. The dependent variable is the reaction times. By conducting this ANOVA, it is possible to obtain the Mean Squares which are required to utilize the formula

$$1 - \frac{(\text{MSparticipants X MSbins})}{(\text{MSbins})}$$

(Neufeld, et al., 2007). The closer to 1 that alpha coefficient is, the more homogenous the groups.

Once group homogeneity is established, it is then possible to calculate capacity ratios and coefficients. The first step is to calculate the observed proportions of respondents in each response time bin for each condition. The next step is to calculate the estimated survivor function [S(t)]. The survivor function indicates the probability that completion has not yet occurred. The survivor function is complementary to the cumulative probability distribution, and as such is calculated as 1 – F(t). The next step is to calculate the integrated hazard function. The formula to calculate the integrated hazard function is

$$H(t)=-\ln[S(t)]$$

where ln(x) stands for the natural logarithm of x. This formula is used in order to calculate the integrated hazard function for capacity ratios and the OR capacity coefficient. However, when calculating the AND capacity coefficient the formula is

$$K(t) = \ln [F(t)]$$

Once integrated hazard functions and K(t) are available, both capacity coefficients and ratios may be calculated. Capacity ratios and coefficients with values less than 1, indicate limited capacity. Values equal to 1 indicate that capacity is unlimited. Values greater than 1 indicate super capacity.

While conclusions can be made regarding capacity requirements based on the value of the capacity ratios or coefficients, currently there is no accepted statistical method to compare whether these capacity measures differ from one another. While traditional ANOVA may include between group or condition post-hoc analysis to test for significant differences, these tests are unavailable for capacity measures. Results will be interpreted based on whether capacity is limited, unlimited or super. Between group differences may be described, however statistical significance is not described. Capacity ratios may also be compared between individuals, if there are an adequate number of response times per individual. The current study, however, does not allow for individual comparisons.

Analysis 1

The purpose of the current analysis is to assess, through the use of capacity ratios and coefficients, whether smoking taxes cognitive capacity amongst groups of smokers. For each of the stimulus configurations, capacity ratios will be calculated. In addition to capacity ratios between conditions, capacity coefficients will be utilized in order to assess the processing requirements of distinct stimulus configurations.

Three sets of capacity ratios will be calculated. The first set will compare the capacity requirements for the smoking condition to the non-smoking condition. For participants with little smoking experience, capacity ratios should be less than one, indicating limited capacity. This would indicate that smoking is capacity taxing in comparison to the non-smoking condition. For regular smokers, the capacity ratio should be close to 1, indicating unlimited capacity. This would support the hypothesis, that smoking is automatic, when using capacity as an indicator of automaticity.

The second set of capacity ratios will compare the pseudosmoking condition to the non-smoking condition. It is hypothesized that the capacity ratios for all groups, regardless of smoking categorization, should be less than one indicating limited capacity. For participants with minimal smoking experience, the act of bringing the cigarette to and from the mouth should require capacity. For experienced smokers, the act of ceasing the smoking behaviour partway through, should tax capacity. If smoking is automatic, than attention is needed in order to cease the behaviour.

The third set of capacity ratios will compare the capacity requirements of pseudosmoking to the smoking condition. For non-smokers, the capacity ratios should be greater than 1, indicating that pseudosmoking is less capacity taxing than smoking. The inhalation of cigarette smoke should be more capacity taxing than simply moving the cigarette to the mouth, so the removal of this behaviour during the pseudosmoking condition may reduce the capacity need. For smokers, the capacity ratio should be less than1, indicating pseudosmoking actually taxes capacity more than smoking itself.

Capacity coefficients will be calculated in order to assess the capacity demand for the OR and AND conditions. The stimulus configurations were designed to investigate mental processing in one and two target conditions. For the OR conditions, the hypothesis is that double targets will tax capacity more in comparison to the single target condition. The stimulus configurations also include double and single non-target conditions (OO, O_, _O). These configurations may be used in order to calculate the AND coefficient, as both stimuli need to be processed in order to make a response. The use of the AND coefficient is currently theoretical and has not been used in research, so its use is largely exploratory.

Method

Participants

114 (70 males, 44 females) individuals participated in the study. Participants were initially recruited from an introductory psychology course and received course credit for their participation. Participants were required to be at least 19 years of age, as that is the legal smoking age in Ontario, where the experiment took place. There were no participatory requirements in terms of smoking experience. Additional participants were recruited by poster advertising and paid $10. Posters were placed throughout a university campus and attracted both students and university staff. These participants were required to smoke at least one cigarette per day. The addition of this inclusion criterion was required to increase the number of regular smokers in the study. Data from 100 participants were used in the analysis as reaction times were unavailable for 14 participants due to a number of reasons, including computer difficulties and refusal to smoke in the smoking conditions. These participants still received course credits or payment for their participation. This study received ethical approval from a university Research Ethics Board, which can be found in Appendix A.

Measures

A number of smoking measures were included in the study in order to obtain information about smoking status, including current smoking behavior, past experience with smoking,

and levels of dependence.

Timeline Follow-back Interview (Sobell & Sobell, 1995). The Timeline Follow-back Interview was originally designed to assess recent alcohol use but has been adapted for other types of substance use (TLFB; Sobell & Sobell). The TLFB uses a calendar in order to assess substance use in a specified timeframe, usually within the last 30 days. There have been some studies that have examined the validity and reliability of the TLFB in measuring smoking. Brown et al. (1998) found that the TLFB had good validity and reliability in assessing smoking patterns in a sample of adults undergoing smoking cessation treatment. Correlations between the TLFB and monitored daily reports of smoking ranged from 0.67 to 0.97. Lewis-Esquerre et al.(2005) also found the TLFB was valid in a sample of adolescent smokers. It was significantly correlated with two measures of smoking dependence, saliva nicotine levels and respiratory symptoms, as well as a global single item measure ("How many cigarettes per day do you currently smoke?"). When participants were completing the TLFB for smoking they were also asked to estimate the number of standard size alcohol drinks (1 bottle of regular strength beer, 1.5 ounces of hard liquor, or 5 ounces of wine) they consumed per day during the last 30 days in order to assess for cigarette and alcohol co-use.

Lifetime Smoking Questionnaire (Baxter & Hinson, 2001). The Lifetime Smoking Questionnaire was specifically designed for Baxter & Hinson's study of the automaticity of smoking behaviour. Participants were asked to estimate when they had their first cigarette and when their most recent cigarette was smoked. They were also asked to estimate the number of cigarettes smoked per day for each year that they had smoked. In their study, Baxter and Hinson found a naturally occurring division among the participants; those who smoked more than 800 cigarettes over their lifetime and those who smoked less than 100 cigarettes in their lifetime. However, Massak (unpublished thesis) found that 11 out of 88 participants did not fall within one of these two groups in a similar sample of undergraduate students.

The Fagerstrom Test for Nicotine Dependence (FTND; Heatherton, Kozlowski, Frecker, & Fagerstrom, 1991). The FTND is a 10-item measure in which participants are queried about their smoking habits. Scores on the FTND range from 0-10, with higher scores indicating more severe levels of dependence. Although the FTND is one of the most widely used assessment instruments to measure dependency, it does have limitations. It has been criticized for its psychometric properties, and its assumption that physical dependence is the key component in dependence (Piper et al., 2004).

Nicotine Dependence Syndrome Scale (NDSS; Shiffman, Waters, & Hickcox, 2004). In addition to an overall score of dependence, the questionnaire includes five subscales measuring various aspects of dependence. These subscales are Drive, Priority, Tolerance, Continuity and Stereotypy. Drive measures the compulsion to smoke. Priority reflects the desire to smoke above other activities. Tolerance measures the diminished effects of smoking over time. Continuity measures the regularity of smoking (i.e. smoking patterns over the day). Stereotypy measures the invariance of smoking behaviour (i.e. smoking is not affected by mood or illness). The scale is scored to produce standardized scores with a mean of 0, and a standard deviation of 1 (Shiffman & Sayette, 2005). This measure has been found to have high internal reliability and good test-retest correlations.

Task

As previously described, the purpose of the task was to respond by pressing one keyboard key when the target "X" was shown on the stimulus screen, and to respond by pressing a different keyboard key if "X" was not present. Responses were made by using the non-dominant hand, so that the dominant hand was available to hold a cigarette during the smoking conditions. Right handed smokers were asked to press the "A" key if the target was present and the "S" key if the target was absent. Left-handed smokers were asked to press the "K" key if the target was present and the "L" key if the target was absent. The experimental task was set up using e-prime, from which the reaction times were obtained. The task took place in a well-ventilated laboratory that had been designed and constructed to conduct smoking research.

After providing consent to participate and completing the previously described questionnaires, participants completed practice trials to become accustomed to the task. They received 80 practice trials, with 10 presentations of each of the 8 stimulus configurations. These practice trials were completed under no smoking conditions. After this practice period there were 20 presentations of each of the 8 stimulus configurations. under each smoking condition. The stimulus configurations were presented in random order.

Each participant completed the task under each of the smoking conditions. One condition was the no-smoking condition. All smoking paraphernalia was out of view of the participant during this condition, and they were instructed to complete the reaction time task. Another condition was the smoking condition, in which the participant was asked to do the monitoring task while smoking a cigarette. Participants were given the option of smoking their own cigarettes or presented with three brands of cigarettes supplied by the experimenter. These brands were the top sellers at a university convenience store according to the store clerk. Participants were asked to light the cigarette prior to beginning the task. Participants were instructed to smoke at a pace under which they would normally smoke. A clean ashtray was provided for each of the conditions. The final condition was the pseudosmoking condition which involved holding a lit cigarette and bringing it up to the mouth, without inhaling. Participants were instructed to light the cigarette prior to beginning the task, and to bring the cigarette to their mouth without "taking a drag." Participants were supplied with a clean ash tray in order to shake off the excess ash.

The smoking conditions were presented randomly to each participant. The experimenter remained in the room with the participant during all of the conditions to ensure that instructions regarding the smoking conditions were followed. Conditions were presented consecutively, with no breaks between conditions due to time constraints. The completion of questionnaires and the reaction time task took approximately 45 minutes. Participants were debriefed following the completion of the experiment.

Results

Smoking Groups

Participants were categorized as belonging to one of four groups based on their smoking in the last 30 days as measured by the TLFB: no smoking in the last 30 days, smoking on 1-14 days in the last 30 days (light smokers), smoking on 16-29 days in the last 30 days (moderate), and smoking everyday in the last 30 days. The light smokers and moderate smokers were a naturally occurring group, as no participants smoked on 15 days. Descriptive information can be found in Table 1. There were significant differences between groups in terms of demographics, smoking history, smoking behaviour in the last 30 days, and on dependence measures. There was a significant difference in mean age between groups F (3, 96) = 3.51, p = 0.018, and post hoc analysis revealed that the daily smokers were significantly older than light smokers. There was also a significant difference between the number of years since trying the first cigarette F (3, 95) = 6.07, p = .001. The daily smokers had their first cigarette significantly earlier than the other three groups.

There was a significant difference between smoking groups on the Fagerstrom Test of Nicotine Dependence F (2, 72) = 14.43, p < .001. Daily smokers had significantly higher dependence scores than the light and moderate smokers. There was also a significant difference between groups on the NDSS- Total Score F (2, 68) = 29.52, p > 0.001. Daily smokers had significantly higher scores than both the light and moderate smokers, and the moderate smokers had higher scores than the light smokers. On the subscales of the NDSS, there were significant differences between groups on two of the subscales. An alpha value of .0125 was used to determine significance (.05/4). There were significant differences on the Drive scale, F (2, 69) = 24.85, p < .001, with daily smokers scoring higher than both the light and moderate smokers and the moderate smokers scoring higher than the light smokers. There were also significant differences within the Continuity subscale, F (2, 69) = 4.84 p = .011, with daily smokers scoring higher than the other two groups.

Table 1.
Means and standard deviations of smoking group descriptives.

	Non-Smokers (N=20)	Light Smokers (N=25)	Moderate Smokers (N=29)	Daily Smokers (N=26)
% Female	40.0%	28.0%	41.4%	53.8%
Age	20.20	19.59	20.38	23.69
	(2.31)	(1.33)	(2.27)	(9.21)
# of years since	3.60	3.38	5.00	8.96
first cigarette	(3.75)	(2.08)	(2.94)	(8.96)
Dependence Measures				
FTND		0.11	1.10	2.38
		(0.32)	(1.65)	(8.96)
NDSS – Total		-2.16	-1.15	-0.34
		(0.67)	(0.87)	(0.72)
NDSS – Drive		-2.48	-1.43	-0.35
		(0.86)	(1.15)	(0.90)
NDSS –		-0.40	-0.07	0.22
Stereotypy		(0.97)	(0.85)	(0.85)
NDSS-		-1.38	-1.23	-0.44
Continuity		(1.15)	(1.08)	(1.12)
NDSS- Priority		-0.41	-0.47	-0.67
		(0.27)	(0.45)	(0.61)
NDSS -		-1.00	-0.42	-0.18
Tolerance		(0.80)	(1.16)	(1.17)
Timeline Followback				
# of smoking		6.16	24.93	30.00
days		(4.03)	(4.11)	(0)
# of cigarettes		17.04	136.97	252.38
		(19.62)	(114.00)	(122.34)
# of cigarettes		2.24	5.18	8.41
per day		(1.44)	(3.89)	(4.08)
Maximum # of		4.56	10.52	14.23
cigarettes		(4.24)	(7.27)	(6.30)

There were a number of differences in smoking behavior in the last 30 days as measured by the Timeline Followback. As would be expected given how the groups were formed, there were between group differences in number of days smoked in the last 30 days, F (2, 77) = 358.44, $p < .001$. There was also a significant difference in total number of cigarettes smoked in the last 30 days, F (2, 77) = 36.37, $p < .001$, with each group differing significantly from each of the others. The groups each differed significantly from one another when comparing daily cigarette consumption on smoking days, F (2, 77) = 21.04, $p < .001$. There were also significant differences between groups on maximum number of cigarettes per day, F (2, 77) = 16.06, $p = .011$. Daily smokers had a significantly higher maximum consumption than did the light smoking group.

Reaction Time Data

Reaction time data were available for 100 participants. Participants accrued 160 reaction times for each smoking condition, which were composed of 20 reaction times for each of the eight stimulus configurations. Reaction times from all participants were combined for each stimulus type in each of the three smoking conditions. Combining the reaction times resulted in a possible 2000 reaction time data points per stimulus configuration in each of the conditions. Reaction times for inaccurate responses were removed from the dataset. Of 48 000 reaction times, there were 1930 inaccurate responses (4%), and these were discarded. This is well below the 10% guideline that is suggested by Wenger and Townsend (2007).

Reaction times less than 100 ms and greater than 3000 ms were discarded. There were 16 response times that were less than 100ms, and 212 that were greater than 3000 ms. An estimate of movement time (160 ms) was subtracted from each reaction time. Negative values were set to zero (n=31). Residual reactions times for each type of data presentation in each of the three conditions were binned. The bin values were 0-400 ms, 400-600 ms, 600-800 ms, and 800 -1000 ms, which were adapted from Neufeld et al., (2007).

Capacity Ratios Between Conditions

To assess whether capacity was affected based on smoking condition and smoker groups, capacity ratios were calculated. As reaction times were to be analyzed based on smoking group memberships, alpha coefficients were calculated to ensure group homogeneity. This was calculated using the formula

$$1 - \frac{(\text{MS participants X bins})}{(\text{MSbins})}$$

These values can be found in Table 2. All alpha coefficients were in the 0.99 range, indicating good group homogeneity. In order to calculate capacity ratios, the observed proportion of reaction times per bin, estimated survivor function and integrated hazard function were calculated for each of the smoking groups in each of the three conditions. These can be found in Tables 1-12 in Appendix B.

Capacity ratios for each of the stimulus configurations can be found in Figures 4- 11. Overall, results suggest that smoking is capacity taxing regardless of smoking experience. Regardless of smoking group, the capacity ratios for smoking in comparison to non-smoking for all stimulus configurations were less than 1. This indicates that smoking does limit capacity, which supports the hypothesis that smoking does require cognitive capacity. Capacity ratios for those with the least amount of smoking experience, the non-smokers, ranged from 0.52-0.73 in the 0-400 bin, 0.52-0.68 in the 400-600 bin, 0.46-0.65 in the 600-800 bin, and 0.44-0.62 in the 800-1000 bin. For light smokers the capacity ratios ranged from 0.48-0.82 in the 0-400 ms bin, 0.46-0.67 in the 400-600 ms bin, 0.46-0.63 in the 600-800 ms bin, and 0.44-0.68 in the 800-1000 ms bin. For both non-smokers and light smokers, capacity ratios remained relatively stable across time bins, with slight decreases in capacity ratios in later time bins.

Table 2

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Coefficient Alpha Calculation for Smoking Groups X Bins

		MS bins *participants	MS bins	Coefficient alpha
XX				
	No smoking	7645.83	3535749.83	0.9978
	Smoking	1725.32	10458058.00	0.9998
	Pseudosmoking	2450.27	11345521.98	0.9998
X _				
	No smoking	7124.15	5660248.09	0.9987
	Smoking	5427.08	11570786.69	0.9995
	Pseudosmoking	7066.94	15398417.62	0.9995
_X				
	No smoking	6942.09	6974099.03	0.9990
	Smoking	6746.85	12255822.96	0.9994
	Pseudosmoking	8195.26	12858634.90	0.9994
XO				
	No smoking	5741.33	7661772.41	0.9993
	Smoking	7810.19	13795751.56	0.9994
	Pseudosmoking	2738.72	13468934.69	0.9998
OX				
	No smoking	1024.36	12944877.65	0.9999
	Smoking	2707.79	16096144.32	0.9998
	Pseudosmoking	3551.76	16724572.33	0.9998
OO				
	No smoking	4339.58	6467303.59	0.9993
	Smoking	4510.68	9768719.13	0.9995
	Pseudosmoking	5509.47	13089817.53	0.9996
O_				
	No smoking	3361.36	7963541.14	0.9996
	Smoking	2180.09	12879103.90	0.9998
	Pseudosmoking	7516.30	13879912.98	0.9995
_O				
	No smoking	4288.73	8518582.25	0.9995
	Smoking	2590.30	11588595.49	0.9998
	Pseudosmoking	3665.47	15381487.46	0.9998

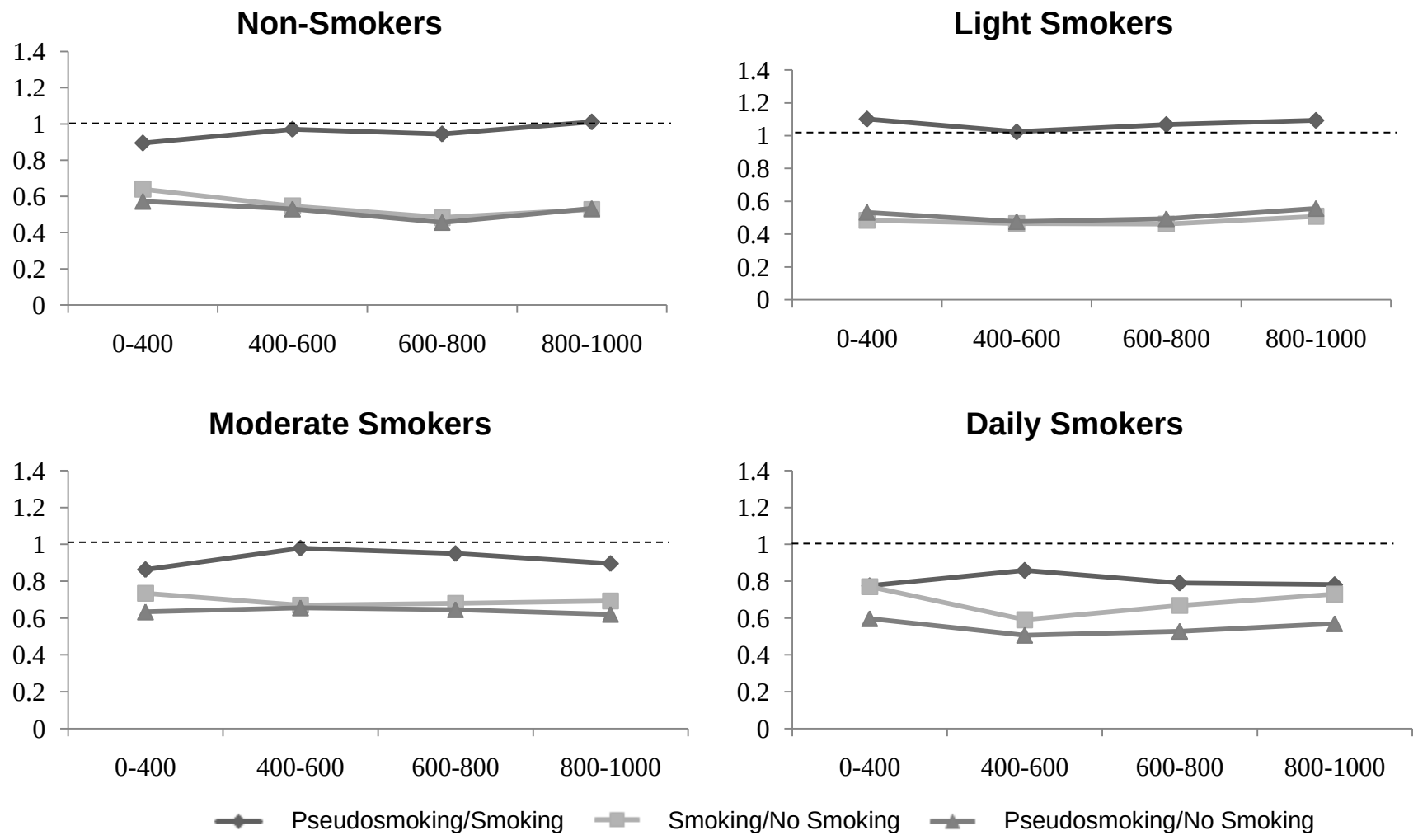

Figure 4. Capacity Ratios for XX Configuration

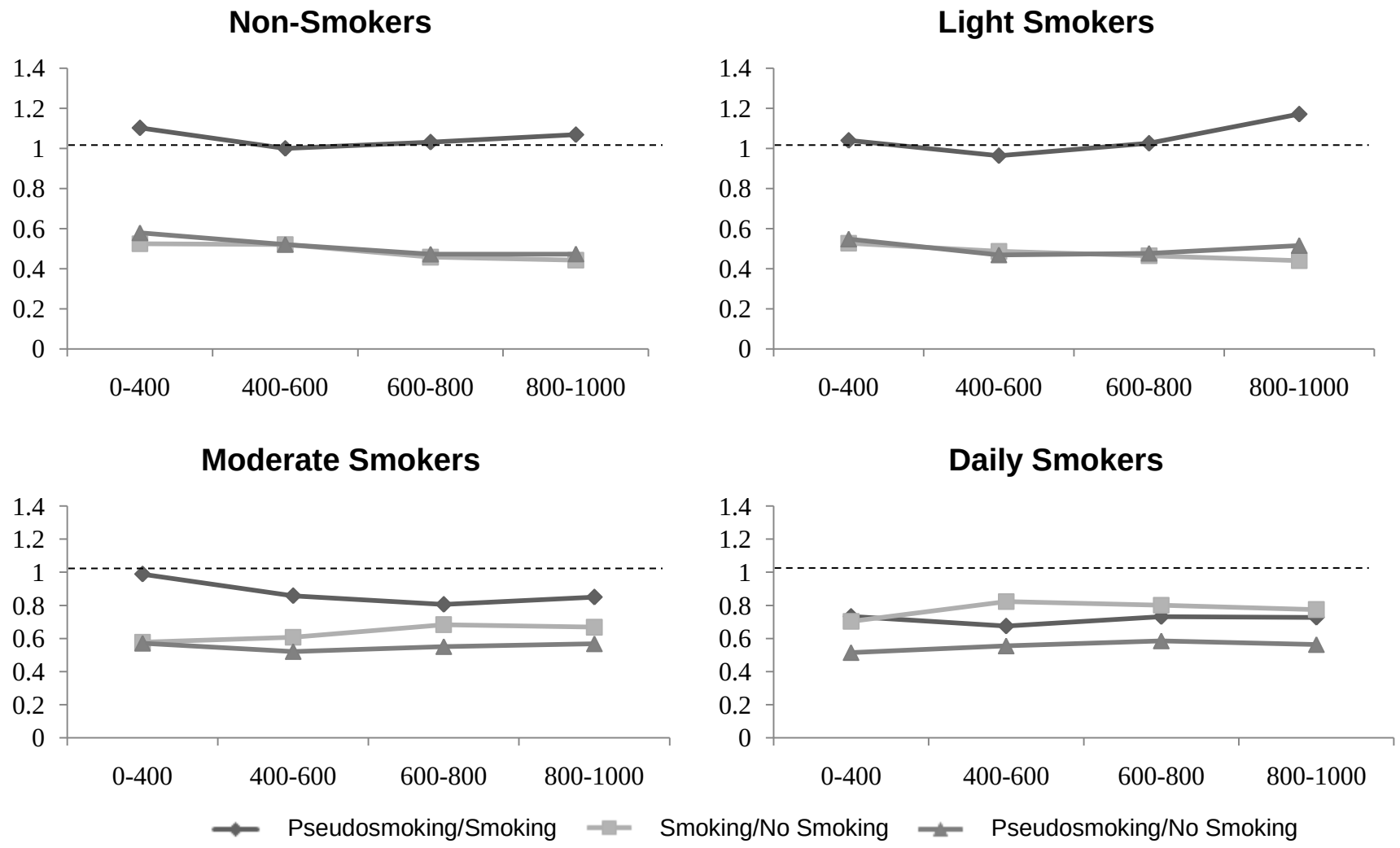

Figure 5. Capacity Ratios for X_ Configuration

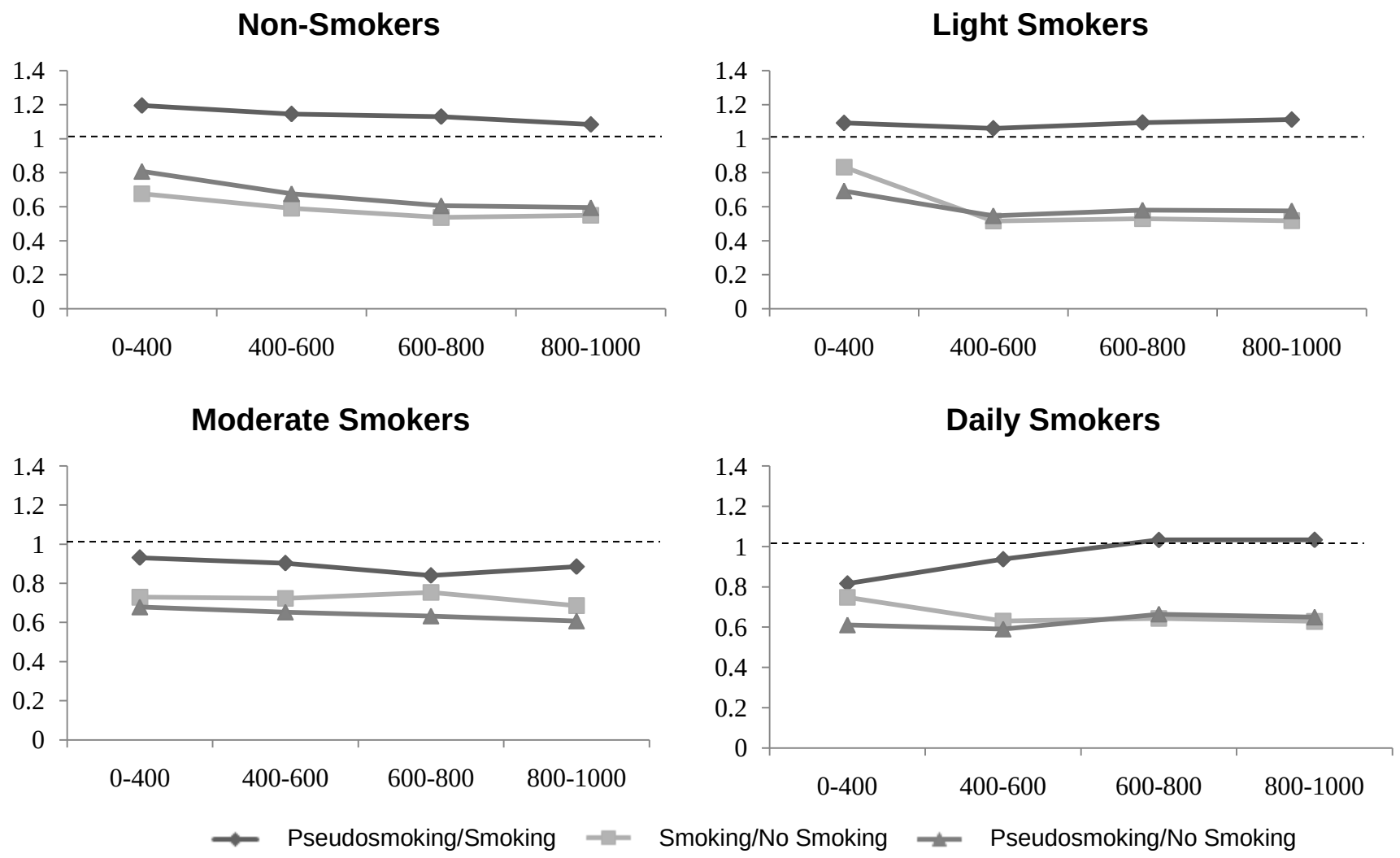

Figure 6. Capacity Ratios for _X Configuration

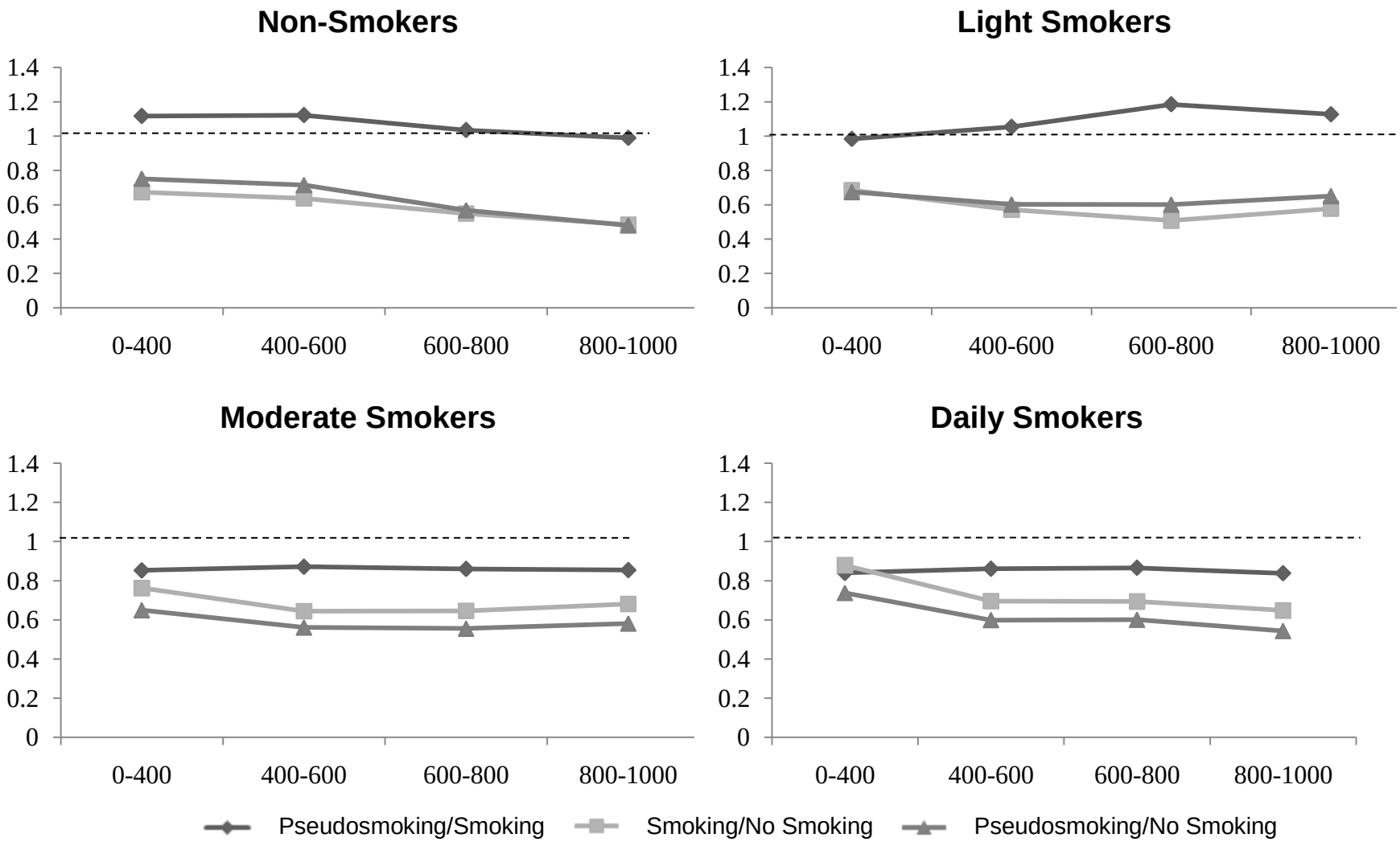

Figure 7. Capacity Ratios for XO Configuration

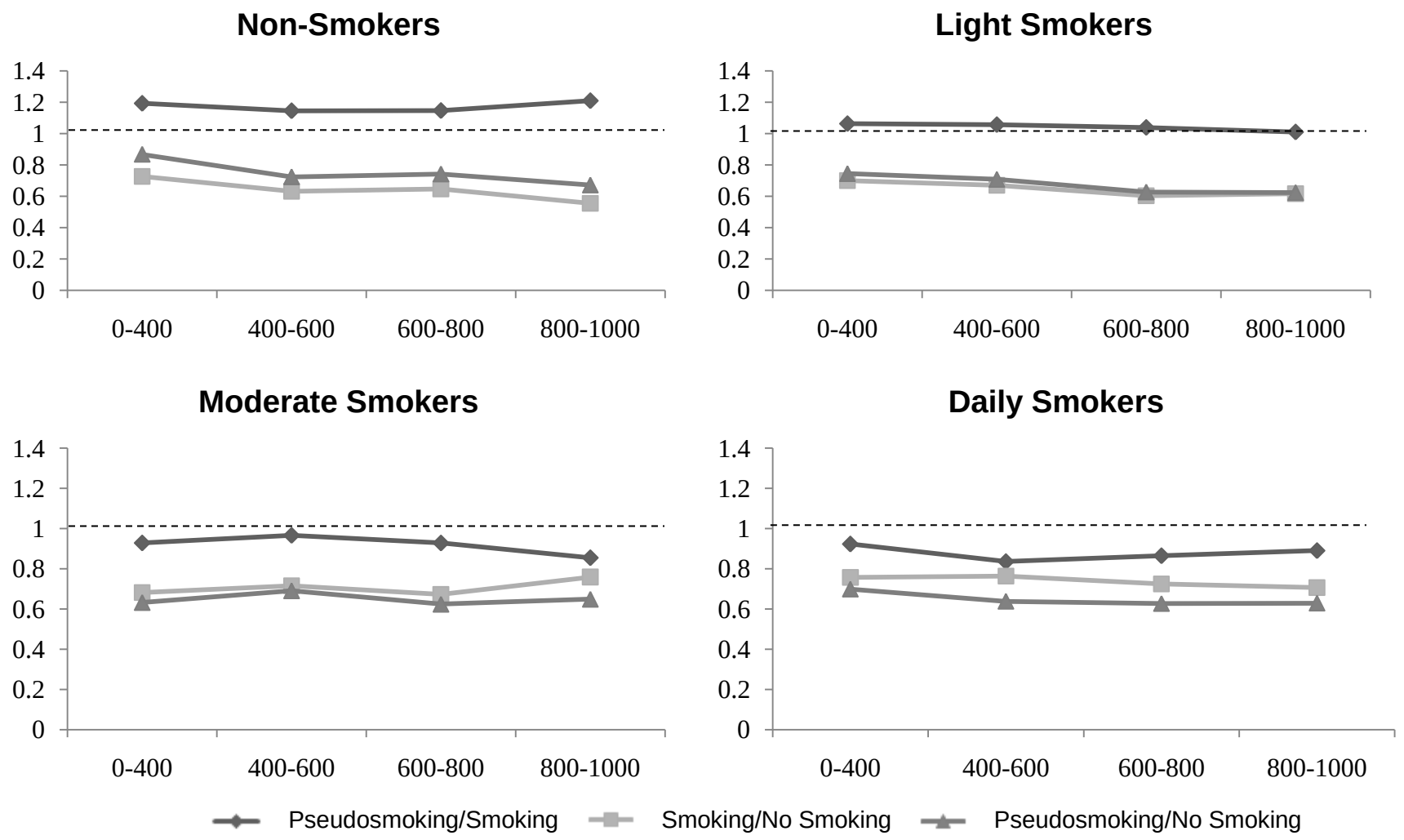

Figure 8. Capacity Ratios for OX Configuration

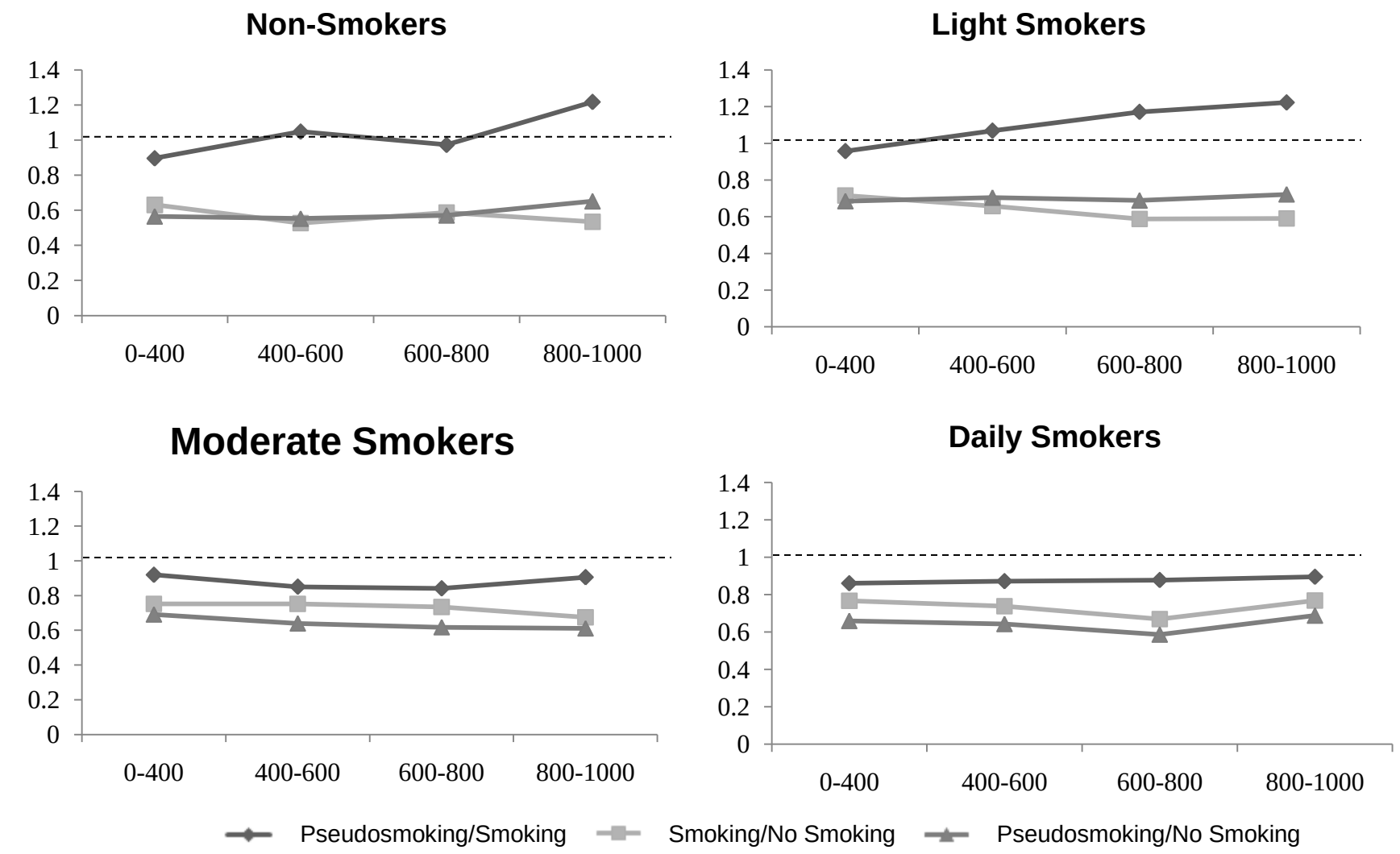

Figure 9. Capacity Ratios for OO Configuration

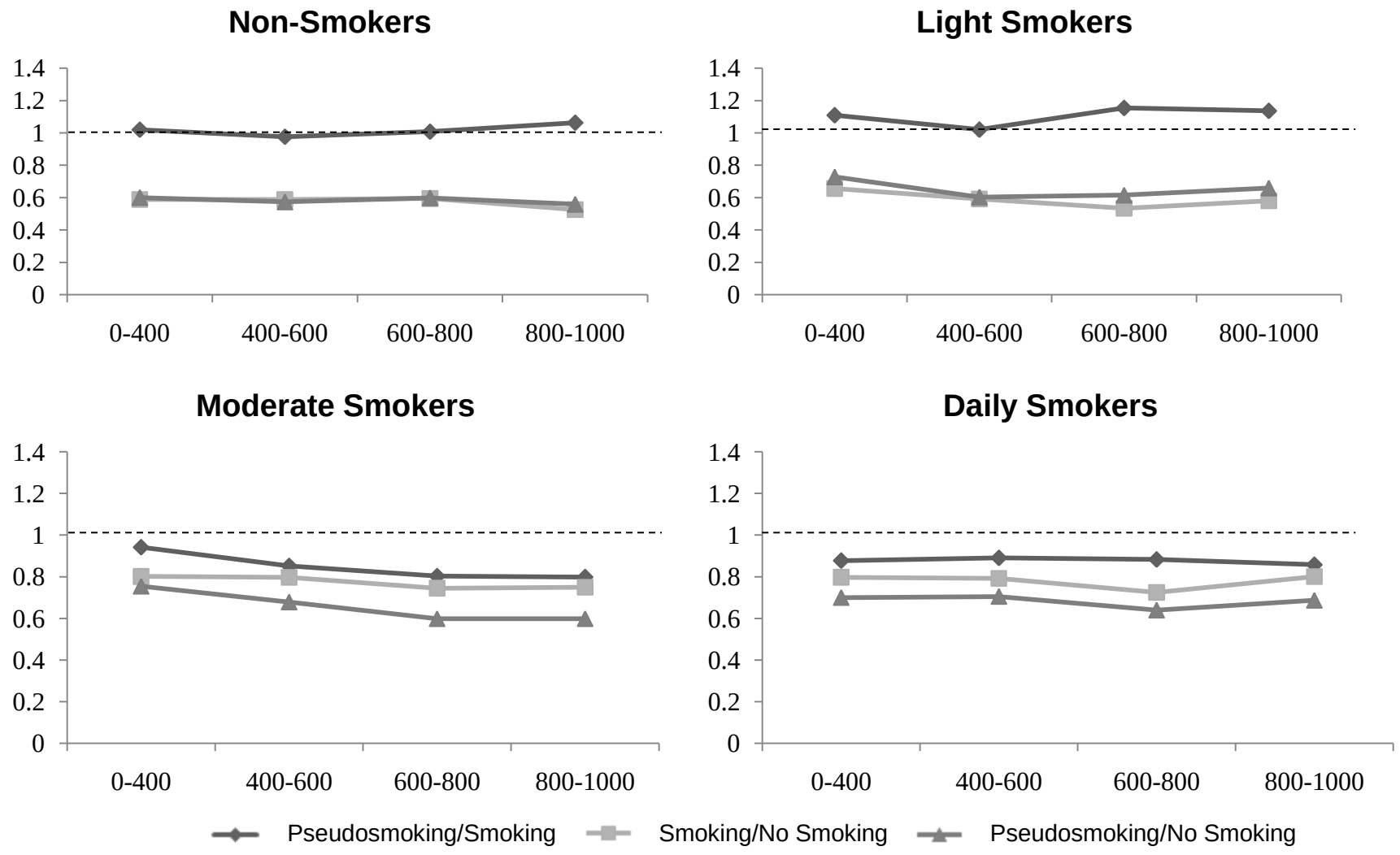

Figure 10. Capacity Ratios for O _ Configuration

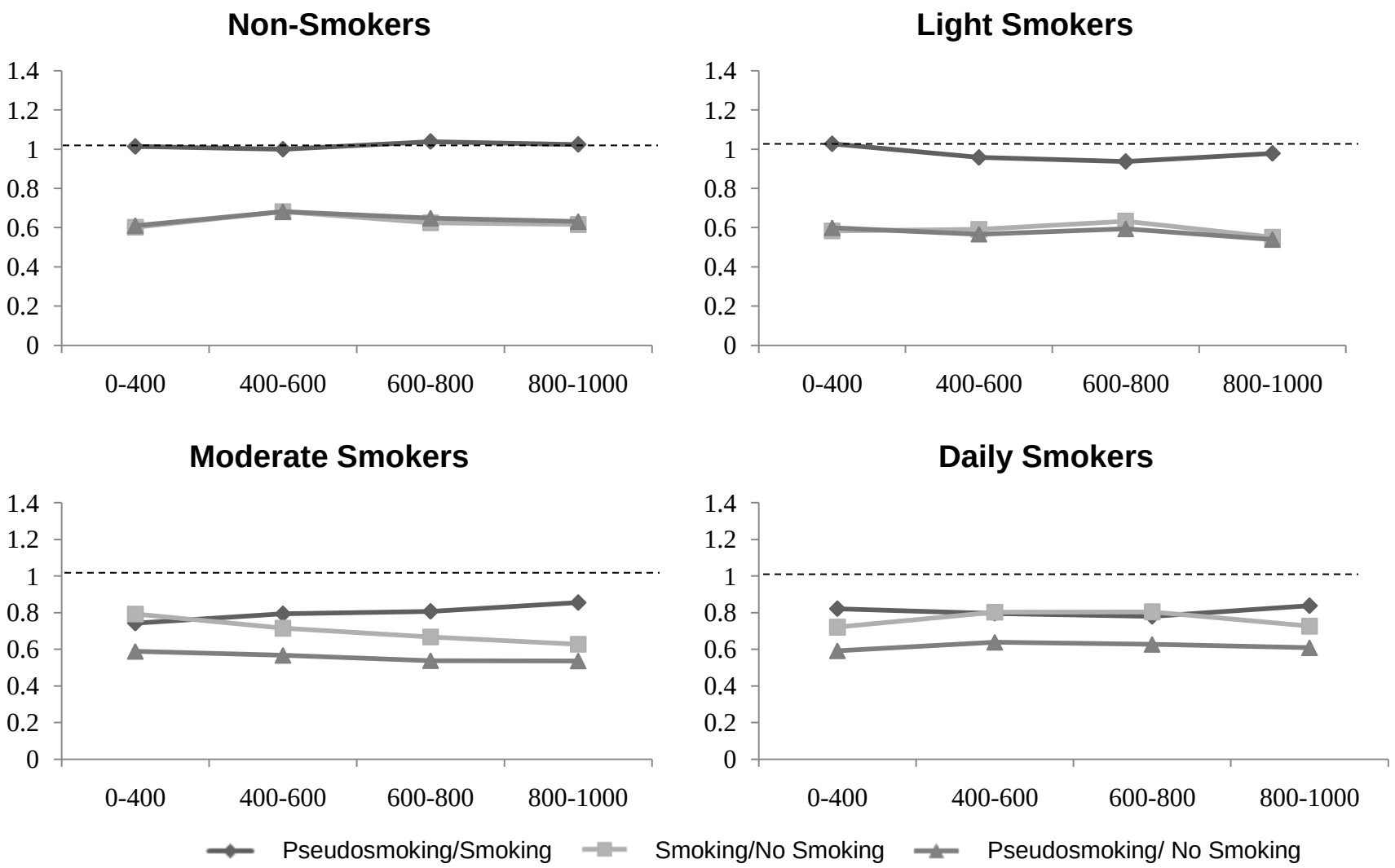

*Figure 11.*Capacity Ratios for _O Configuration

The capacity ratios comparing the smoking and nonsmoking conditions in moderate and daily smokers were consistently below 1. These results did not support the hypothesis that smoking had become automatic in the smokers in the present study using capacity as an indicator of automaticity. The capacity ratios for moderate smokers ranged from .58-0.80 in the 0-400 ms bin, 0.61-0.80 in the 400-600 ms bin, 0.65-0.75 in the 600-800 ms bin, and 0.63-0.76 in the 800-1000 ms bin. For the daily smokers the capacity ratios ranged from 0.70-0.88 in the 0-400 ms bin, 0.5-0.82 in the 400-600 ms bin, 0.64-0.80 in the 600-800 ms bin, and 0.63-0.80 in the 800-1000 ms bin. As with the non- and light smokers, capacity ratios tended to slightly decrease as response times increased, however these decreases were relatively small.

Pseudosmoking also taxed capacity in comparison to the non-smoking condition, as all of the capacity ratios comparing the two conditions were less than one. This supported the hypothesis that pseudosmoking would be capacity taxing for all smoking groups. For non- smokers the capacity ratios ranged from 0.57-0.87 in the 0-400 ms bins, 0.52-0.72 in the 400-600 ms bins, 0.46-0.74 in the 600-800 ms bin, and 0.47-0.67 in the 800-1000 ms bin. For light smokers, the capacity ratios ranged from 0.53-0.74 in the 0-400 ms bin, 0.47- 0.71 in the 400-600 ms bin, 0.48-0.69 in the 600-800 ms bin, 0.52-0.72 in the 800-1000 ms bin. These capacity ratios tended to overlap with the ratios comparing smoking and no smoking. For moderate smokers, the capacity ratios ranged from 0.57-0.75 in the 0-400 ms bin, 0.52-0.69 in the 400-600 ms bin, 0.54-0.65 in the 600-800 bin, 0.54-0.65 in the 800-1000 ms bin. For daily smokers the capacity ratios ranged from 0.52-0.75 in the 0-400 ms bin, 0.51-0.71 in the 400-600 ms bin, 0.53-0.64 in the 600-800 ms bin, and 0.54-0.69 in the 800-1000 ms bin. These capacity ratios tended to be lower than the capacity ratios comparing smoking and no smoking conditions.

When comparing capacity requirements for pseudosmoking and smoking, results did differ depending on smoking group. For non-smokers and light smokers, the majority of capacity ratios were consistently above 1, indicating that processing during the pseudosmoking condition was less capacity taxing than in the smoking conditions. For non-smokers, the capacity ratios ranged from 0.89-1.19 in the 0-400 ms bins, 0.97-1.15 in

the 400-600 bin, 0.94-1.15 in the 600-800 ms bin, and 0.99-1.22 in the 800-1000 ms bin. For light smokers, capacity ratios ranged from 0.96-1.11 in the 0-400 ms bin, 0.96-1.07 in the 400-600 ms bin, 0.94-1.18 in the 600-800 ms bin, and 0.98-1.22 in the 800-1000 ms bin. These ratios were considerably higher than the capacity ratios comparing smoking or pseudosmoking to nonsmoking. For moderate smokers capacity ratios ranged from 0.74-0.99 in the 0-400 ms bin, 0.79-0.98 in the 400-600 ms bin, 0.80-0.95 in the 600-800 ms bin, and 0.80-0.91 in the 800-1000 ms bin. For daily smokers, capacity ratios ranged from 0.73-0.93, 0.67-0.94 in the 400-600 ms bin, 0.73-1.03 in the 600-800 ms bin, 0.73-1.03 in the 800-1000 ms bin. These capacity ratios were also higher than the other two sets of capacity ratios.

General Capacity Coefficients

The reaction times for the non-smoking condition for all of the participants were used to calculate capacity coefficients. The observed proportion of reaction times per bin, estimated survivor function and capacity indices needed for the calculation of the OR and AND capacity coefficients can be found in Tables 13-22 in Appendix B.

The OR and AND capacity coefficients are plotted in Figure 12. For the OR condition, the capacity coefficient indicates limited capacity when two targets are presented in comparison to only one target being presented. Mental processing is taxed more when two targets are presented simultaneously than would be expected based on the sum of the two targets presented individually. The presence of redundant targets not only does not enhance processing, it impedes it. These coefficients were relatively stable across time. There were minimal differences in the standard OR coefficient

$$C_o(t) = \frac{H\,x,x(t)}{(H\,x,\_\,(t) + H\,\_,x(t))}$$

and the coefficient incorporating the distracters

$$C_o(t) = \frac{H\,x,x(t)}{(H\,x,o\,(t) + H\,o,x(t))}$$

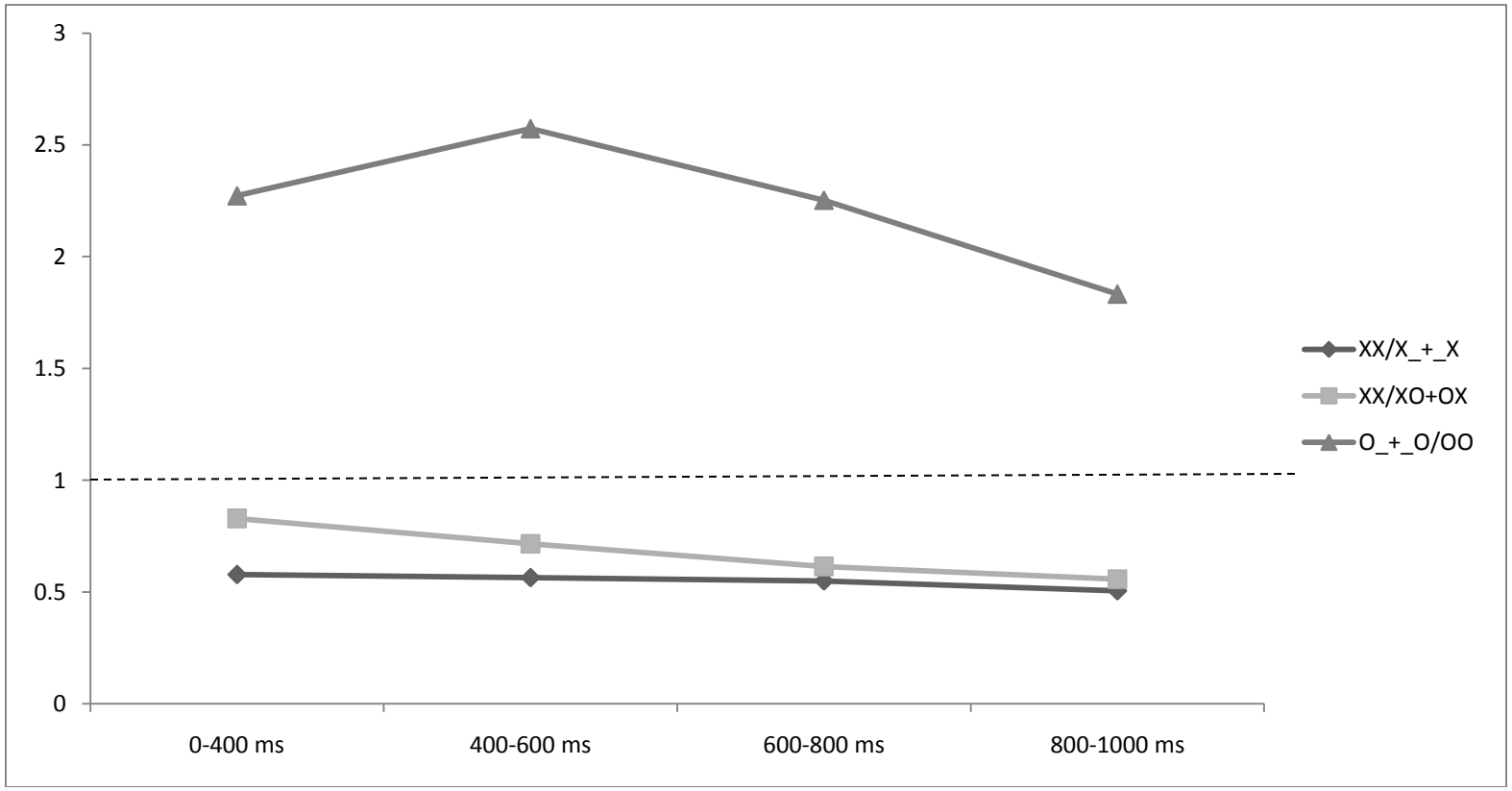

Figure 12. AND and OR Coefficients for Complete Sample

There was a slightly higher capacity coefficient in the first bin when distracters were present.

The AND coefficient was greater than 1, indicating super capacity. Redundant non-targets enhanced mental processing in comparison to the sum of the individual non-targets. The coefficients peaked in the second time bin, and decreased over time. This indicates that the greatest OO advantage was not immediately seen, but rather in the later responders. However, this advantage decreased when additional time lapsed.

The OR capacity coefficients using the standard formula for the all of the smoking groups in the smoking and pseudosmoking conditions can be found in Figure 13. The coefficients were all less than 1, indicating limited capacity. The non-smokers had the highest coefficients consistently in the smoking condition. The coefficient values for the pseudosmoking condition were similar across all of the groups. These results indicated that regardless of smoking group or condition, the double targets XX were capacity taxing in comparison to the individual targets. The OR coefficients using the alternate formula can be found in Figure 14. For the smoking condition, the non-smokers had larger coefficient values in the first two time bins, but these differences disappeared in the later time bins. In the pseudosmoking condition, the moderate smokers had consistently higher coefficients than the other groups in all but the first bin.

The AND coefficients for the smoking and pseudosmoking condition can be found in Figure 15. All of the coefficients were greater than 1, indicating super capacity. In the smoking condition, the light smokers had the highest coefficients in all but the first time bin. There were no distinct patterns among the other groups. In the pseudosmoking condition, the light smokers had capacity coefficients similar to the other groups in the first time bin, however the values increased as reaction times elapsed.

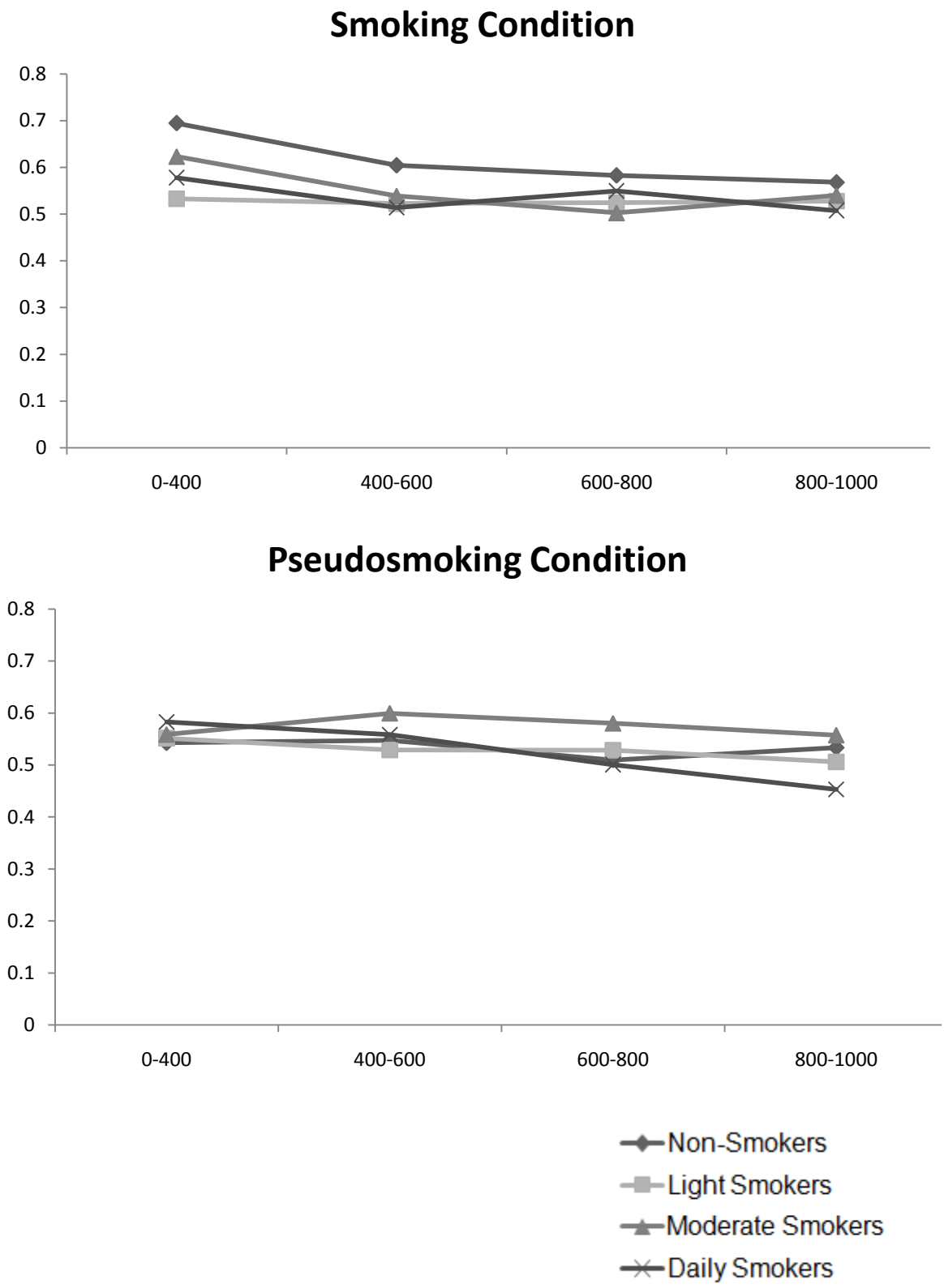

Figure 13. OR Coefficient (standard formula)

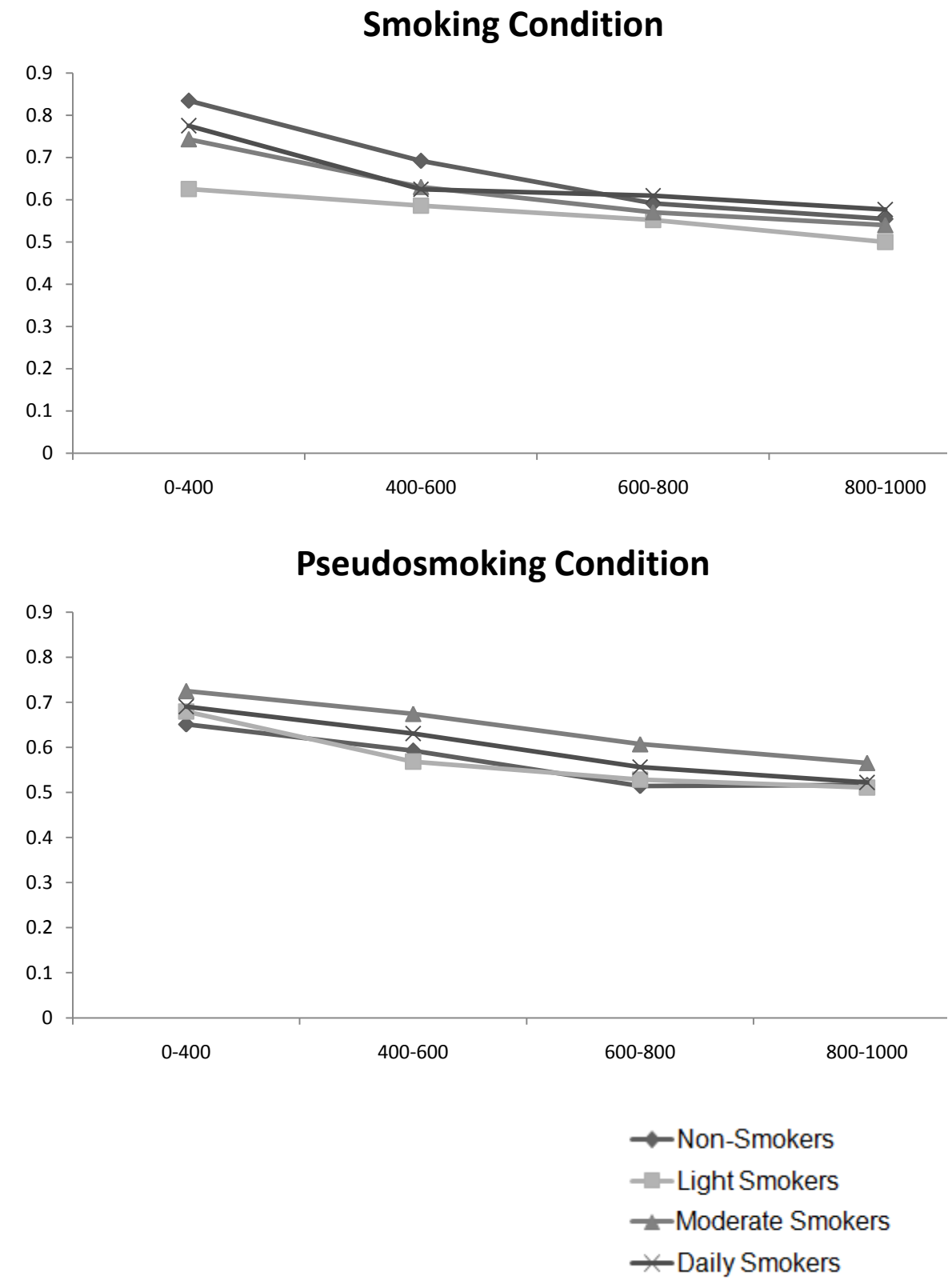

Figure 14. OR Coefficient (alternate formula)

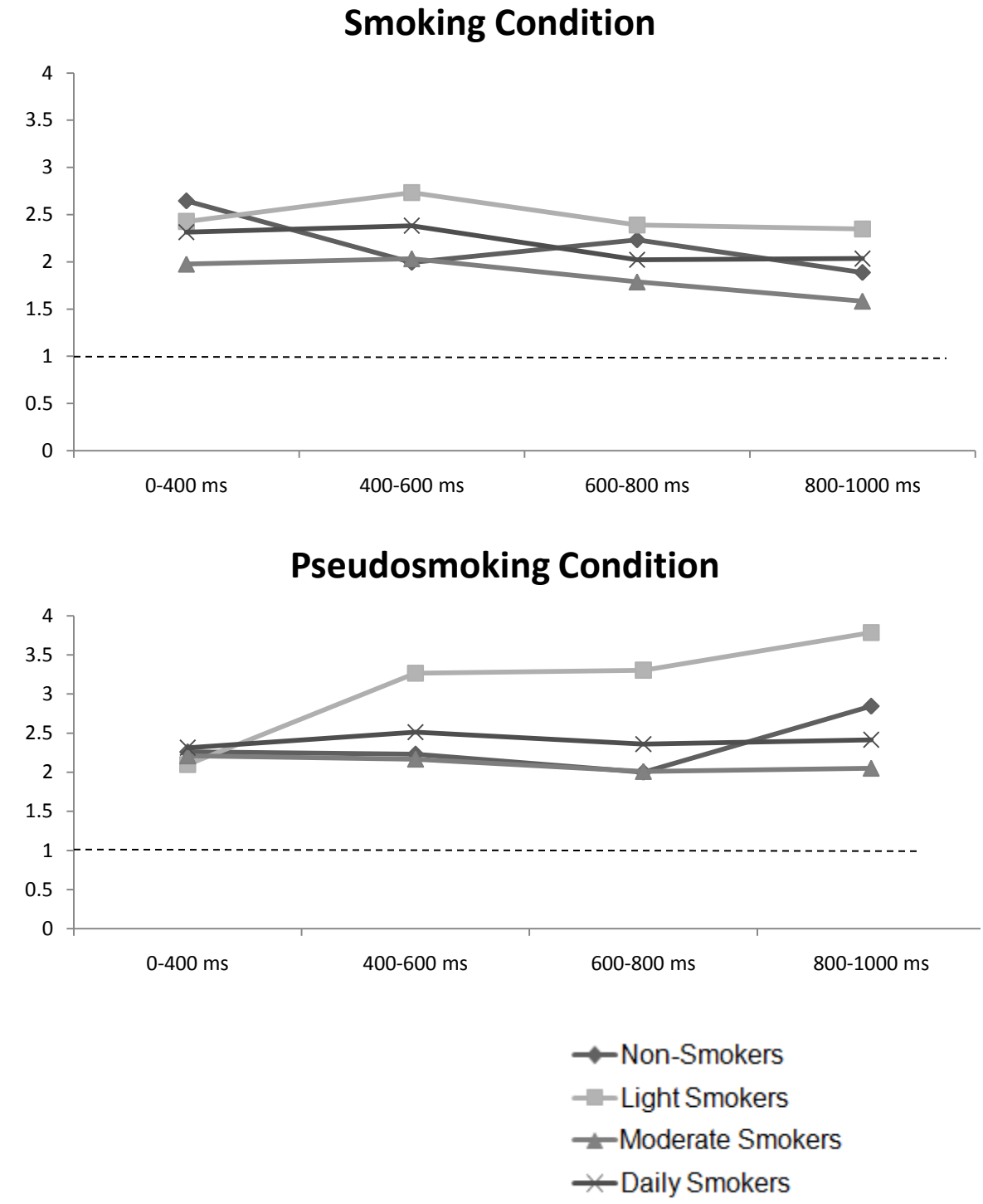

Figure 15. AND Coefficient

Discussion

The purpose of the current analysis was to examine whether smoking taxed capacity during a simple response task, and whether capacity was taxed differently for individuals with varying levels of smoking experience. Capacity was defined as a feature of automaticity, in a pure insertion sense: the addition of smoking behaviour should not impact the processing of a secondary task. Capacity ratios comparing the smoking and non-smoking conditions indicated that smoking does tax capacity, regardless of smoking status. However, the capacity requirements appear to decrease with smoking experience. If capacity is accepted as an indicator of automaticity, then the hypothesis that smoking becomes automatic was not supported. Daily smokers in the current sample required cognitive capacity in order to complete smoking behaviour.

Despite the finding that smoking does tax cognitive resources among experienced smokers, it may be premature to declare that smoking does not become automatic. One of the limitations of the current analysis is the relatively low dependence levels of the daily smokers. Dependence levels as measured by the FTND were low, and lower than in the average smoker as measured by the NDSS. Daily cigarette consumption was also relatively low. The average daily cigarette consumption of the daily smokers was 8.7 cigarettes per day. Smoking less than 10 cigarettes per day is often classified as "light" smoking across other studies (Repetto, Caldwell, & Zimmerman, 2005). It would be beneficial to replicate the current analysis using heavier smokers to investigate whether the capacity requirements for the smoking condition compared to the non-smoking condition would indicate unlimited capacity. This lack of heavy smokers is a definite limitation.

Within the current sample, capacity ratios increased as smoking experience and dependence levels increased, so it is possible that that capacity ratios would continue to increase in a more dependent, heavier smoking sample. Further research with a sample including heavier smokers could clarify whether capacity ratios comparing smoking and non-smoking do eventually reach 1, or whether there is an upper limit for capacity ratios

with respect to smoking behavior.

Pseudosmoking also taxed capacity for all smoking groups. For non-smokers and light smokers, the act of having a lit cigarette to manage while doing the search task was capacity taxing. While the participants did not have to inhale the smoke, the act of bringing the cigarette to and from the mouth was cognitively taxing. Based on these results alone, it is impossible to state why all groups of smokers found pseudosmoking to be cognitively taxing. It could be argued that for both non- and light smokers, pseudosmoking is a novel behaviour and that is the reason why it is cognitive taxing. The novelty of smoking behaviour, regardless of whether smoke needed to be inhaled or not, was sufficient to tax capacity.

For moderate and daily smokers, it is possible that pseudosmoking is cognitively taxing because they have to stop a previously learned behaviour. Smoking is a frequent behaviour for these groups, and in order to cease the behaviour partway cognitive resources may be required. The capacity ratios comparing pseudosmoking to no smoking were consistently lower than the capacity ratios comparing smoking to no smoking across all stimulus configurations. This suggests that pseudosmoking is more cognitively taxing than smoking. There is something about pseudosmoking that taxes capacity, but it would be premature to definitively conclude that the act of ceasing smoking behaviour itself is cognitive taxing. However, examining the capacity ratios comparing pseudosmoking and smoking conditions provides a direct comparison of smoking behaviour that either includes or excludes inhalation of smoke.

While pseudosmoking was found to be capacity taxing for all smoking groups in comparison to the non-smoking conditions, the most telling differences were found across capacity ratios comparing the pseudosmoking and smoking conditions. This set of capacity ratios were the only ones where there was a clear difference between smoking groups. For the non- and light smokers, the majority of capacity ratios were greater than 1, indicating super capacity.

Capacity ratios were less than 1 for moderate and daily smokers. These differences

between groups suggests that the act of smoke inhalation may be a key behavioural component of smoking in terms of mental processing. While bringing the cigarette to and from the mouth is capacity taxing, as indicated by limited capacity when comparing pseudosmoking and no smoking, the act of smoke inhalation requires even more cognitive processing. This finding suggests that smoking behaviour can be conceptualized as having not only distinct observable behavioural components, but also distinct components that require different types of cognitive processing.

Whereas extra cognitive work is required in order to inhale smoke in inexperienced smokers, extra cognitive effort is required for experienced smokers in order to stop the smoking schema and prevent the inhalation of smoke . The act of ceasing the smoking behaviour partway requires cognitive effort, as illustrated by the capacity ratio being less than 1, when comparing pseudosmoking to smoking. If capacity is accepted as an indicator of automaticity, this suggests that it is the inhalation of smoke that may become automatic in experienced smokers, and preventing this behaviour requires controlled processing.

The discussion above suggests that it may be simplistic to conceptualize smoking as a single behavior. As it has been suggested that the study of automatic processing should be a features-based approach, it may be that the study of smoking itself should be a features based approach. As the capacity ratios comparing cognitive processing for the pseudosmoking and smoking conditions suggest, there may be different cognitive requirements for the motor components of smoking (bringing the cigarette to and from the mouth), and the inhalation of the smoke. From a strictly behavioural perspective, arm movements in order to bring the cigarette to and from the mouth may require a different type of cognitive processing than inhalation of smoke.

In terms of the stimulus configurations, it was found that double targets (XX) did tax capacity in comparison to the sum of the individual targets for all smoking groups in all of the smoking conditions. This is consistent with Townsend and Wenger's (2001) study on faces, where they found that the processing of two facial features was more

cognitively taxing than the processing single features. Additional information does not seem to help mental processing, but rather taxes it. In contrast the AND coefficient indicated super capacity. The processing of two non-targets was more efficient than the processing of the sum of each non-target presented individually. The presence of additional information appeared to help participants decide that no target was present more efficiently than the sum of the two individual targets. This may indicate that targets and non-targets are processed in different ways. The double targets may be processed in a Gestalt manner, in which the double targets are processed as a whole, rather than as two individual targets.

There was little variation between groups when examining OR capacity coefficients for the smoking and pseudosmoking conditions. The non-smokers tended to have the highest coefficient values in the smoking condition, indicating that the double target was less capacity taxing in comparison to single targets, than for the other groups. This may suggest that the effect of the stimulus load is tempered by the experimental condition. The capacity ratios comparing smoking to non-smoking tended to be the lowest for non-smokers in comparison to the other smoking groups. Since smoking was already capacity taxing, the capacity requirements of stimulus configurations did not have as large an effect. It is possible that there is a limit to how much capacity is taxed. The OR coefficients in the pseudosmoking condition were relatively consistent across smoking groups.

The AND coefficients in the smoking and pseudosmoking condition indicated that it was the light smokers who found the double non-targets presentation most beneficial. The capacity coefficient was consistently higher for the light smokers in all but the first time bin, in both the smoking and pseudosmoking conditions. It is unclear why the light smokers would benefit more greatly in comparison to the other groups.

Another interesting finding was that the X on the left hand of the screen appeared to be processed more efficiently than the X on the right hand of the screen when examining the individual H(t) values (can be seen in Table 13 in Appendix B). This difference was also

found for the XO and OX configuration. One possibility to explain this finding is that reading in Western culture goes from left to right, and participants may have simply been "reading" the target positions serially. However, Simgasiewicz et al. (2010) found that regardless of culture, there is a left visual field advantage when doing a visual search task. They argue that the left field advantage is due to organizational processes between the two brain hemispheres. The left visual field advantage was not found when the stimulus target was the non-target, O. The O on the right side of the screen was processed faster than when the O was presented on the left side of the screen.

Regardless of the general processing requirements, the pattern of results was consistent across each of the stimulus configurations for each of the smoking groups. This finding may suggest that the complexity of the secondary task does not appear to have an impact on capacity. It is possible that regardless of what a smoker is doing, cigarette smoking still requires the same level of mental processing. When considering the number of things that smokers do while smoking, such as driving a car, this may have some serious implications. If mental processing is required in order to smoke, it is possible that this reduces the availability of mental processing for other tasks. Of course this is speculative at this point, but it may be beneficial to conduct studies to test the impact of smoking on other behaviours.

Overall, the current analysis has provided evidence that smoking does require cognitive processing, and that certain components of smoking may be more cognitively demanding than others. Inhalation of smoke, in particular, seems to demand mental processing in non- and light smokers. For moderate and daily smokers, however, it is the cessation of this behaviour that requires additional capacity. A feature-based approach to the study of smoking behaviour may be beneficial.

Analysis 2

Townsend & Wenger (2004a) argue that much of the evidence for psychological theory is circumstantial and can only be said to be consistent with an assumption versus discerning those assumptions. Traditional statistical analysis, such as analysis of variance, may be viewed as most often involving such an indirect test of assumptions. Mean reaction times may be affected by a number of different factors, which may or may not be due to the variables of interest. By relying on traditional statistical analysis, conclusions are based on the independent variables that are manipulated and the dependent variables that are outputted. All of the processes between input and output are given limited consideration. At best, extraneous variables are controlled for statistically, or at worst, ignored. Luce (1995) argues that to truly understand a behaviour, researchers must move beyond simply looking at inputs and outputs of behaviour, and open the "black box."

Another problem with traditional statistical analysis is that it often assumes that data are normally distributed. The more common distributions for reaction times are positively skewed, with the majority of responses at the front end of the curve. Reaction times at the tail end of the curve are often treated as outliers and discarded. However, these late responses may provide valuable data about the processing in question. Mathematical modeling allows for the analysis of reaction times without dependence on the normal distribution. In addition to reliance of the normal distribution, the focus of traditional analysis is the mean, followed by the standard deviation. Reliance on the mean provides limited information when considering reaction times. If the tail end of the curve is discarded, the mean can be greatly impacted. Also, when dealing with hundreds or thousands of reaction times, it seems simplistic to try to capture all the information in a single value.

Given the differences between the two types of analysis, comparing the results obtained from each type may illustrate these differences on a tangible level. As demonstrated in Analysis 1, smoking did tax capacity, even in the most experienced smokers in the sample. While capacity demands decreased as the level of smoking experience increased,

there was still a cognitive demand during smoking behavior. Given that capacity is being utilized as an indicator of automaticity, the conclusion drawn was that smoking was not fully automatic even in the most experienced smokers in the sample. This is in contrast to conclusions reached by Baxter and Hinson (2001), who used analysis of variance. The purpose of the current analysis is to re-analyze the data from Analysis 1 using repeated measures analysis of variance, and to compare the results between the two analyses. This provides a direct comparison of the two types of analysis, as the same participants and data are used for both.

It is hypothesized that there will be a significant interaction between smoking group and smoking condition. It is predicted that post hoc tests will reveal that non-smokers will have significantly slower reaction times in the smoking and pseudosmoking conditions in comparison to the non-smoking conditions. Daily smokers will have significantly slower response times in the pseudosmoking condition than in the non-smoking condition. Reaction times should be equivalent in the smoking and no-smoking condition. The results for the light smokers should be similar to those of non-smokers, whereas moderate smokers should be similar to daily smokers.

Method

Methodology for obtaining data has been described in Analysis 1. As with the previous analysis, reaction times were amalgamated into one variable according to stimulus configuration and smoking condition (e.g., the XX stimulus configuration under no smoking condition). There were 2000 possible reaction times for each stimulus configuration. Reaction times for inaccurate responses were removed from the data set (N=1930). To ensure that the same reaction times were included as in the previous analysis, reaction times less than 100 ms and greater than 3000 ms were removed from the dataset. However, 160 ms was not subtracted for base processes as this is not typically done in a traditional ANOVA. Smoking group classification (non-smoker, light smoker, moderate smoker, and daily smoker) was kept consistent from Analysis 1.

Repeated measures ANOVA were conducted for each type of stimulus configuration separately with smoking group as the between-group factor and smoking condition as the within-subject factor. Although, as noted above, reaction times were positively skewed the data were not transformed in any manner in order to maintain consistency with the study of Baxter & Hinson.

Results

Mean reaction times and standard deviations for each smoking condition can be found in Table 3. A smoking group by smoking condition interaction was found for all of the stimulus configurations; *F* values can be found in Table 4. The Greenhouse-Geisser adjustment needed to be used for all of the analysis as Mauchly's test of sphericity was significant for all of the analyses. Graphical representation of these interactions can be found in Figure 16.

Post-hoc tests were conducted to compare mean reaction time for pairs of smoking conditions within smoking groups using the Tukey's Honestly Significant Test, e.g., to compare the mean RT for no-smoking to smoking in non-smokers. The statistical program POST-HOC was used. Significant *q* values may be found in Table 5. For all of the smoking groups for each of the stimulus configurations, reaction times in the non-smoking condition were faster than in either the smoking or pseudosmoking conditions. For non-smokers and light smokers, there were no significant differences between the pseudosmoking and smoking conditions for any type of stimulus configurations. For the moderate and daily smokers there were significant differences for some of the stimulus configurations between the pseudosmoking and smoking conditions, and in each instance reaction times were faster in the smoking condition rather than the pseudosmoking condition.

Table 3.

Mean and Standard Deviations of Reaction Times for All Smoking Groups

	Non-Smokers	Light Smokers	Moderate Smokers	Daily Smokers
XX				
No Smoking	430.33 (208.58)	424.10 (132.59)	433.56 (185.53)	423.10 (132.18)
Smoking	543.34 (378.41)	569.37 (388.04)	512.53 (296.14)	467.21 (183.41)
Pseudo-Smoking	542.92 (310.92)	552.37 (341.15)	544.21 (353.68)	515.04 (269.21)
X_				
No Smoking	424.88 (198.08)	408.79 (127.06)	418.14 (168.33)	412.43 (127.06)
Smoking	596.34 (450.36)	555.81 (383.39)	526.17 (328.19)	468.23 (243.92)
Pseudo-Smoking	582.01 (400.58)	542.77 (355.57)	566.93 (387.32)	534.12 (332.64)
_ X				
No Smoking	488.99 (211.72)	474.40 (171.02)	478.14 (180.15)	468.23 (124.98)
Smoking	631.14 (396.77)	588.22 (345.85)	559.27 (283.34)	531.03 (249.96)
Pseudo-Smoking	601.91 (393.72)	571.58 (336.76)	590.30 (359.75)	540.96 (211.61)
XO				
No Smoking	451.99 (157.56)	458.39 (172.43)	474.54 (198.43)	463.36 (143.10)
Smoking	594.26 (409.60)	563.73 (340.79)	555.10 (334.35)	502.33 (219.76)
Pseudo-Smoking	559.44 (326.16)	537.85 (272.04)	612.29 (377.03)	551.38 (313.75)
OX				
No Smoking	545.27 (221.74)	534.42 (198.60)	514.94 (198.15)	531.18 (202.20)
Smoking	678.42 (392.42)	652.90 (396.76)	601.85 (308.34)	592.52 (274.95)
Pseudo-Smoking	643.53 (367.65)	629.94 (326.52)	633.16 (374.55)	603.32 (268.27)

Non-Smokers	Light Smokers	Moderate Smokers	Daily Smokers	Non-Smokers
OO				
No Smoking	503.72 (186.56)	488.86 (161.93)	509.32 (184009)	481.68 (151.66)
Smoking	622.62 (387.79)	590.82 (355.16)	575.35 (303.701)	530.50 (202.70)
Pseudo-Smoking	605.74 (310.70)	576.91 (335.62)	617.80 (389.68)	582.15 (299.92)
O_				
No Smoking	517.25 (200.04)	505.92 (164.72)	526.41 (173.51)	506.04 (148.86)
Smoking	659.94 (378.58)	629.95 (364.51)	588.42 (317.26)	550.98 (257.40)
Pseudo-Smoking	652.92 (355.96)	612.56 (351.08)	617.20 (344.96)	596.09 (315.84)
_O				
No Smoking	510.96 (207.54)	466.80 (132.70)	488.24 (193.09)	476.86 (185.78)
Smoking	645.17 (384.16)	590.41 (357.63)	556.68 (327.92)	530.21 (246.62)
Pseudo-Smoking	642.05 (386.59)	579.46 (313.27)	628.30 (387.35)	572.55 (299.95)

Table 4.

F Values for Smoking Group X Smoking Condition Interactions

Stimulus configuration	*F* value	*p* value
XX	F (5.5, 3164.39) = 3.18	0.005
X_	F (5.34, 3179.26) = 3.70	0.002
_X	F (5.60, 3230.62) = 2.27	0.038
XO	F (5.57, 3266.35) = 5.06	0.001
OX	F (5.76, 3286.49) = 2.14	0.049
OO	F (5.49, 3209.37) = 2.37	0.032
O_	F (5.55, 3162.21) = 3.18	0.005
_O	F (5.43, 3144.41) = 3.28	0.005

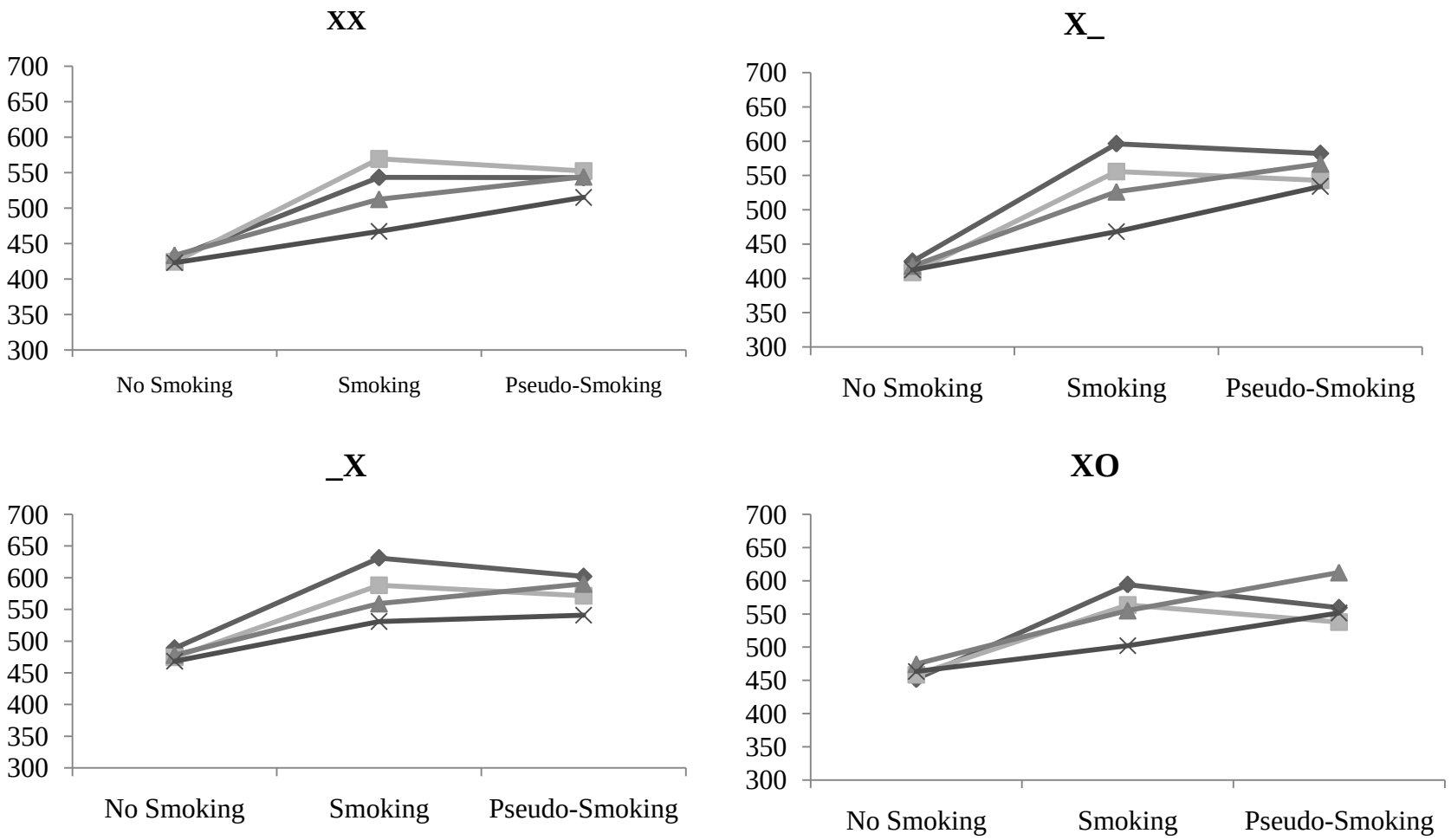

Figure 16. Smoking Group by Condition Interaction

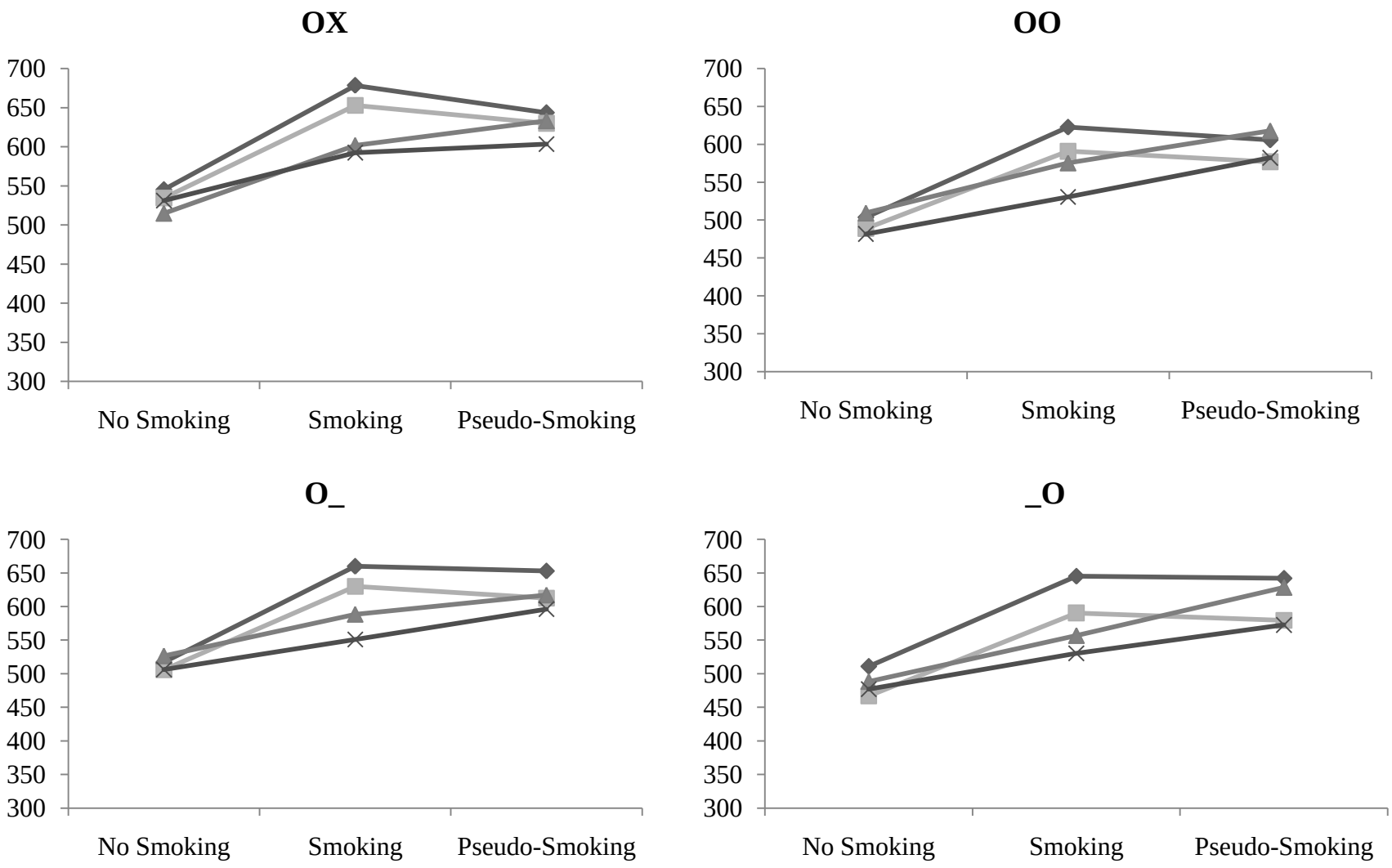

Figure 16. Smoking Group by Condition Interaction

Table 5.

Significant Q-values for Simple Main Effects

	Non-Smokers	Light Smokers	Moderate Smokers	Daily Smokers
No Smoking vs. Smoking				
XX	7.55	7.76	6.44	3.46
X_	10.50	10.03	7.966	3.92
_X	9.91	8.55	6.57	4.89
XO	9.85	8.06	6.59	3.11
OX	8.56	8.69	6.90	4.64
OO	7.93	7.53	5.26	3.75
O_	9.07	8.91	6.98	3.36
_O	8.56	8.78	5.27	3.85
No Smoking vs. Pseudosmoking				
XX	7.58	6.86	9.03	7.21
X_	9.63	9.14	10.97	8.54
_X	7.87	7.30	9.12	5.66
XO	7.44	6.08	11.27	7.01
OX	6.32	7.01	9.38	5.45
OO	6.81	6.51	8.64	7.73
O_	8.63	7.67	4.78	6.73
_O	8.36	8.00	10.78	6.90

	Non-Smokers	Light Smokers	Moderate Smokers	Daily Smokers
Smoking vs. Pseudosmoking				
XX	*ns*	*ns*	*ns*	3.75
X_	*ns*	*ns*	3.01	4.63
_X	*ns*	*ns*	*ns*	*ns*
XO	*ns*	*ns*	4.68	3.89
OX	*ns*	*ns*	2.48	*ns*
OO	*ns*	*ns*	3.38	3.97
O_	*ns*	*ns*	*ns*	3.35
_O	*ns*	*ns*	5.51	3.05

Discussion

The purpose of the current analysis was to compare the conclusions reached based on a traditional analysis of variance of the mean reaction times to the conclusions based on capacity coefficients and ratios of the reaction time data. Overall, the main conclusions reached on the basis of the two types of analyses were similar. Smoking did not appear to be automatic for any of the smoking groups, including the daily smokers. Reactions times were significantly faster in the non-smoking condition than in the smoking condition for all of the groups for all of the stimulus presentation types.
Reaction times for the pseudosmoking condition were significantly slower than the non-smoking condition for all groups for all of stimulus configurations.

In terms of determining whether smoking was automatic the most relevant finding was that for daily smokers, reaction times in the no smoking conditions were significantly faster than in the smoking condition. If smoking were automatic, it would be expected that there would be no difference between the smoking and no smoking condition in these experienced smokers, thus the results did not support the hypothesis. These results are consistent with the previous analysis that the capacity ratios were less than 1 when comparing capacity requirements for the smoking condition in comparison to the no smoking condition. This is not consistent with Baxter & Hinson's (2001) conclusion that smoking was an automatic process.

The main differences between the conclusions reached with the traditional ANOVA and capacity ratios is that the capacity ratios relates to conclusions involving the pseudosmoking and smoking conditions. In Analysis 1, there was a clear difference between groups in capacity ratios comparing pseudosmoking and smoking conditions. Non- and light smokers had capacity ratios greater than 1, whereas the moderate and daily smokers had capacity ratios less than 1. This is direct evidence that the inhalation of smoke was mentally taxing for the less experienced smokers, and the cessation of this behaviour was mentally taxing for more experienced smokers. This suggested that smoking itself may be made up of distinct components, each of which may distinctively

impact capacity. This led to the suggestion in Analysis 1 that smoking may best be studied from a features-based or component approach as compared to viewing it as a unitary behavior.

In the present analysis, there was no evidence of a difference between pseudosmoking and nonsmoking for non- and light smokers. If results were only based on the ANOVA approach, the conclusion would be that performance was similarly impaired in both smoking and pseudosmoking conditions for non- and light smokers. There is no indication that the exclusion of the requirement to inhale smoke was beneficial for these less experienced smokers in terms of mental processing. The comparisons from the present analysis between the smoking and pseudosmoking condition for the moderate and daily smokers were more similar to those using capacity ratios. However, without the comparison of the non- and light smokers, this result is much less informative, and would not have led to any speculation about viewing smoking from a component view, as compared to a unitary view.

Another shortcoming of the ANOVA approach is that there are limitations in the type of post-hoc comparisons that can be made. If an interaction is significant, a researcher must decide whether they want to test for differences between variable A keeping variable B constant, or test for differences between variable B, keeping A constant (Gardner, 2001). Sometimes this choice is clear depending on the research question, however it may be difficult to decide which comparisons are most relevant. By using capacity ratios, it was possible to consider both smoking conditions and smoking groups when making comparisons. As described in the preceding paragraph, the finding that capacity ratios comparing the pseudosmoking and smoking conditions were different between smoking groups provided valuable data that were not present in the ANOVA.

Another difference between the two approaches is the comparison of differences between groups and between conditions. Within traditional analysis, conclusions are based on whether there are significant differences. If the *p* value is significant, the results can be interpreted as different between variables of interest. The mathematical approach does

not tend to take this approach. Conclusions are broader, addressing more general questions such as whether capacity is limited, unlimited, or super. While between group differences can be quite evident, in the case of one group displaying limited capacity and the other showing super capacity, differences between actual capacity ratios are not routinely examined. This difference may be due to the fact that no standard comparisons for capacity ratios have been established. Practically speaking, very few researchers in psychology use these mathematical methods, and there may simply be no demand for such measures. However, this lack of comparison could be frustrating for the traditionally trained researcher in psychology.

A very relevant barrier to the more widespread use of mathematical approaches in comparison to the traditional ANOVA is its perceived inaccessibility. Most researchers in psychology are not exposed to this type of analysis, and it may be relatively daunting to those without a strong background in mathematics. However, as the results of the current studies suggest, findings that are not accessible by traditional statistical methods may remain undiscovered if apprehension about mathematical modeling discourages its use.

Analysis 3

Smoking and alcohol use often occur together (Strine et al., 2005). Smokers are more likely to drink alcohol, and alcohol drinkers are more likely to smoke (Dawson, 2000). This co-occurrence of alcohol drinking and smoking may be a result of numerous factors. There are common factors that increase the risk of alcohol and cigarette use such as parental substance use, peer group use, and availability of drugs and alcohol. The well-established co-use of drugs and alcohol may have implications when considering the cognitive processing involved in smoking behaviour. The presence of alcohol may have an impact on how smoking behaviour develops from a mental processing perspective, due to alcohol's impact on learning.

Alcohol has been found to impair the encoding of new information (Chechile, 2010). If smoking mainly occurs in the presence of alcohol, it is possible that the steps that are required in order to smoke are learned in a less efficient manner than if smoking occurs largely without the co-use of alcohol. This may impact the capacity requirements when needing to enact or inhibit the smoking behavior. High co-users of alcohol and cigarettes may need to engage more cognitive resources than low co-users when smoking.

State dependent learning has been supported for alcohol use (Goodwin et al., 1969; Weingartner, et al., 1976). Information that is learned under intoxication is better recalled under intoxication in comparison to sobriety. Individuals who use cigarettes mostly under the influence of alcohol, may have difficulties smoking while sober. Capacity requirements may be strained to perform a behaviour that is normally performed under alcohol use conditions.

While alcohol itself may have an impact on the learning of smoking behaviour, the consumption patterns of cigarette and alcohol co-users may also have an impact on the learning of smoking behaviour. Individuals who have a strong association between alcohol and cigarette use may have different patterns of cigarette consumption than those who exhibit more independence between the two behaviors. For example, it is lighter,

non-daily smokers who are most likely to smoke only when consuming alcohol (Krukowski et al., 2005). Low-level smokers are more likely to endorse social motives for smoking than heavier smokers (Shiffman et al., 1992; 1994b), and this may also be the case for high alcohol and cigarette co-users who tend to be light smokers. The situations in which high co-users smoke are typically more social, thus smoking in nonsocial contexts may be inconsistent with their usual smoking experience.

Given that alcohol use may impact learning of smoking behaviours, state dependent learning can occur with alcohol, and that light smokers are more likely to be high co-users of alcohol and cigarettes, it is worth investigating whether this relationship does have an impact on capacity ratios. In Analysis 1, light smokers were identified solely on the basis of their cigarette consumption in the previous 30 days. This grouping did not take into consideration alcohol and cigarette co-use. If a large proportion of these light smokers were also high alcohol co-users, then their performance of the reaction time task could have been affected since the task took place under non-alcohol conditions. There may also be differences between moderate and daily smokers with different alcohol and cigarette co-use patterns. However, the smoking behaviour of more regular smokers is less consistently coupled with alcohol, and the absence of alcohol during the reaction time task may be less impactful than would be true for lighter smokers.

The goal of the current analysis is to explore the relationship between cigarette smoking and alcohol use, and its impact on capacity ratios. The main focus is to explore capacity ratios in individuals with equal consumptions patterns, and different alcohol and cigarette coupling patterns. It is hypothesized that a large proportion of light smokers will use cigarettes and alcohol together. If this is the case their performance on the reaction time task should be more greatly impaired, as the smoking is not taking placing under their usual smoking circumstances, i.e. while drinking alcohol. These differences may also occur among heavier smokers who differ in their coupling patterns of alcohol and cigarettes, but since there is more independence between smoking and drinking in regular smokers the effects may be less prominent.

Method

Data from Analysis 1 were used for the current analysis. Alcohol use variables were created from the Timeline Followback Interview. When completing the TLFB calendar participants were asked to indicate both the number of standard drinks they had on each day for the last 30 days, in addition to the number of cigarettes they smoked per day. The number of smoking only days, drinking only days, smoking and drinking days, total smoking days, total drinking days and no smoking or drinking days were calculated and used to identify participants with respect to alcohol/smoking co-use patterns.

Results

Only participants who smoked in the last 30 days were used in the present analysis since it involved smoking and alcohol co-use. Participants who had smoked in the last 30 days were grouped according to the criteria used in Analysis 1: light smokers smoked from 1-14 days, moderate smokers smoked from 16-29 days, and daily smokers smoked every day. The mean number of smoking only, drinking only, smoking and drinking, no drinking and no smoking days can be found in Table 6. Not surprisingly, there were significant differences between groups for all of the variables. Light smokers had a significantly higher number of no smoking/no drinking days than moderate smokers. The daily smokers were not included in this analysis as they had 0 no smoking/no drinking days. There were significant differences between groups on smoking/drinking days. Post-hoc tests using Tukey's HSD indicated that light smokers had significantly fewer of these days than the other two smoking groups. Similarly, the light smokers had fewer smoking/no drinking days than did the other two groups. The light smokers had a higher number of no smoking/drinking days than did the moderate smokers. Again, the daily smokers were excluded from this analysis as they had 0 no smoking/drinking days.

Smokers in the different smoking groups were classified as low or high cigarette and alcohol co-users based on the proportion of days in which they co-used in relation to all smoking days. The number and proportion of smokers in each of these groups can be

Table 6.

Mean Number and Standard Deviation of Co-Use Days for All Smoking Groups

	Light Smokers (*n*=25)	Moderate Smokers (*n*=29)	Daily Smokers (*n*=26)
No Smoking/No Drinking[1]	19.24 (3.89)	3.48 (3.66)	0
Smoking/Drinking[2]	4.68 (3.64)	8.14 (5.03)	7.9615 (4.72)
Smoking/No Drinking[3]	1.44 (1.66)	16.66 (3.65)	22.00 (4.67)
No Smoking/Drinking[4]	3.76 (3.32)	0.69 (1.58)	0

[1] t (52) = 15.33, $p < .001$

[2] F (2,77) = 105.93, $p < .001$

[3] F (2,77) = 23.55, $p < .001$

[4] t (33.24) = 4.23, $p < .001$

found in Table 7. Low co-users were defined as participants who coupled alcohol and cigarettes on 0-40% of all smoking occasions. High co-users were those participants who coupled alcohol and cigarettes on 60-100% of all smoking occasions. This operational definition was most consistent with a naturally occurring separation in the data, maintained a distinct difference between groups, and retained 71 of 80 possible participants. In order to have a sufficient number of reaction times to calculate capacity ratios, moderate and high smokers who were high co-users were combined into a single group. This resulted in five groups: light smokers/low co-user, light smokers/high co-user, moderate smokers/low co-user, daily smokers/low co-user, and moderate and daily smokers/high co-user. Observed bin proportions, estimated survivor functions and integrated hazard functions can be found in Tables 23-37 in Appendix B.

Capacity ratios for the stimulus configurations can be found in Figures 17-24. The graphs in the top panels represent the capacity ratios comparing smoking and no smoking ratios, the graphs in the middle panel represent capacity ratios comparing pseudosmoking and no smoking, and the bottom panels represent capacity ratios for the pseudosmoking/smoking conditions.

Across stimulus configurations, capacity ratios comparing smoking and no smoking were less than 1, indicating that smoking was capacity taxing. This is consistent with the results of Analysis 1. Overall, the light smokers/high co-users tended to have the lowest capacity ratios, indicating that smoking was most capacity taxing for this group. The light smokers/low co-users had higher capacity ratios, overall, than did the light smokers/high co-users. There were no striking differences between the moderate smokers and daily/low co-users and the moderate and daily smokers/high co-users except in the X_ configurations. The high co-users had the second lowest capacity ratios consistently, whereas the daily smokers/low co-users had the highest.

Table 7.

Number and Proportion of Low and High Cigarette and Alcohol Co-Users Among Smoking Groups

	Low co-users	High co-users
Light Smokers	5 (20%)	19 (72%)
Moderate Smokers	20 (72.4%)	5 (13.8%)
Daily Smokers	21 (80.8%)	1 (3.8%)

Note: For purposes of analysis the moderate and daily high co-users were combined into a single group.

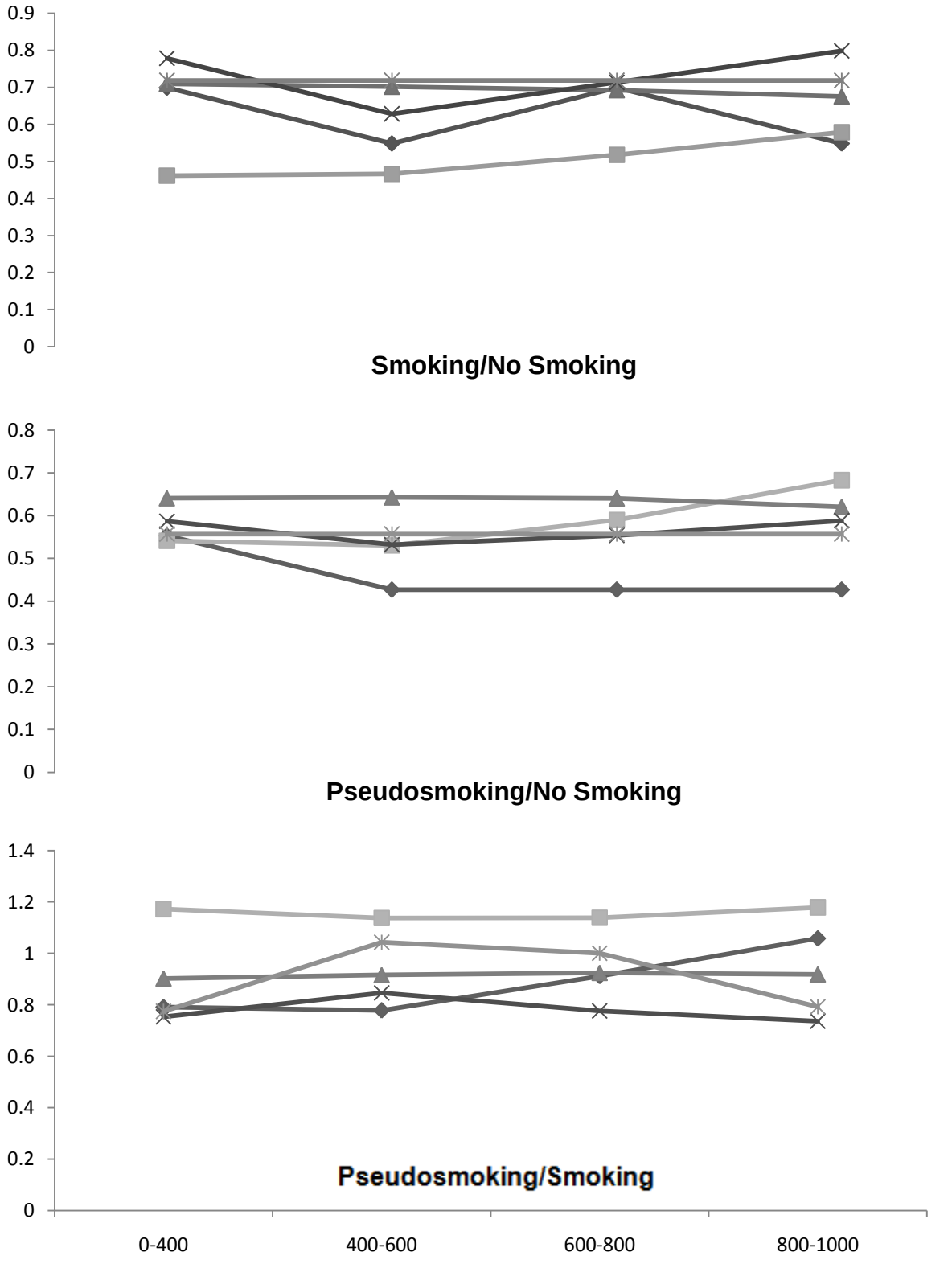

Figure 17. Capacity ratios for XX configuration

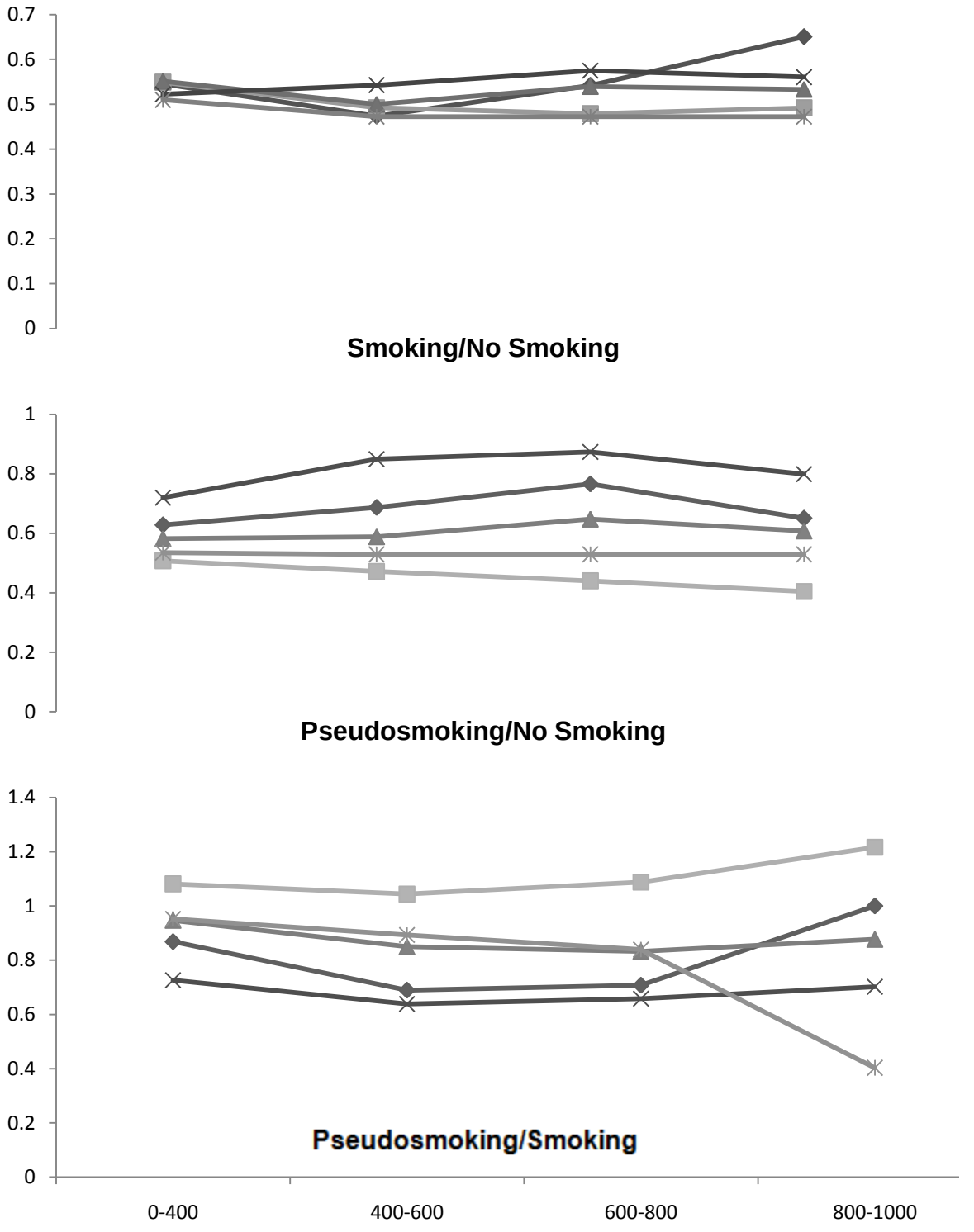

Figure 18. Capacity Ratio for X_ Configuration

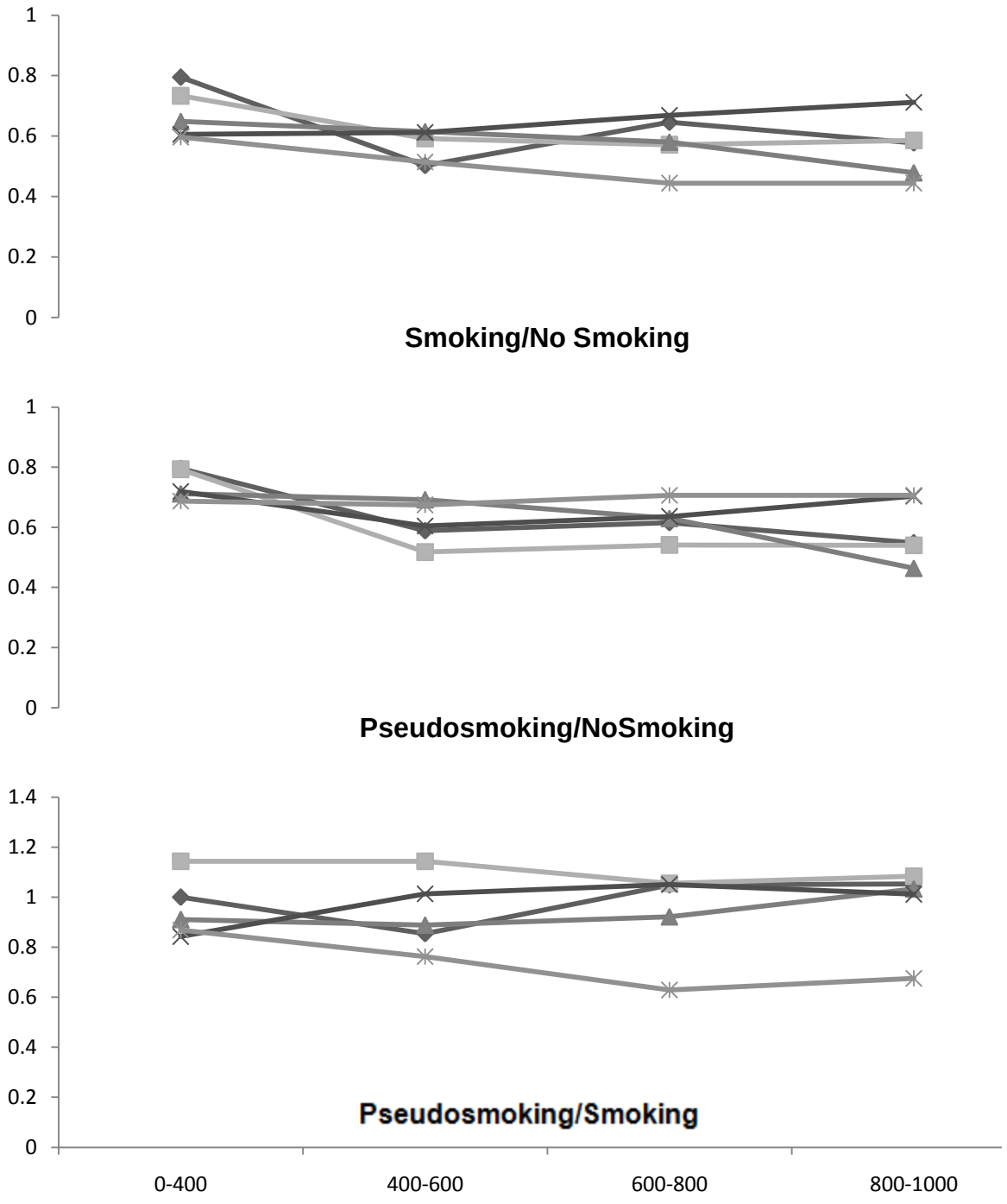

Figure 19. Capacity Ratio for _X Configuration

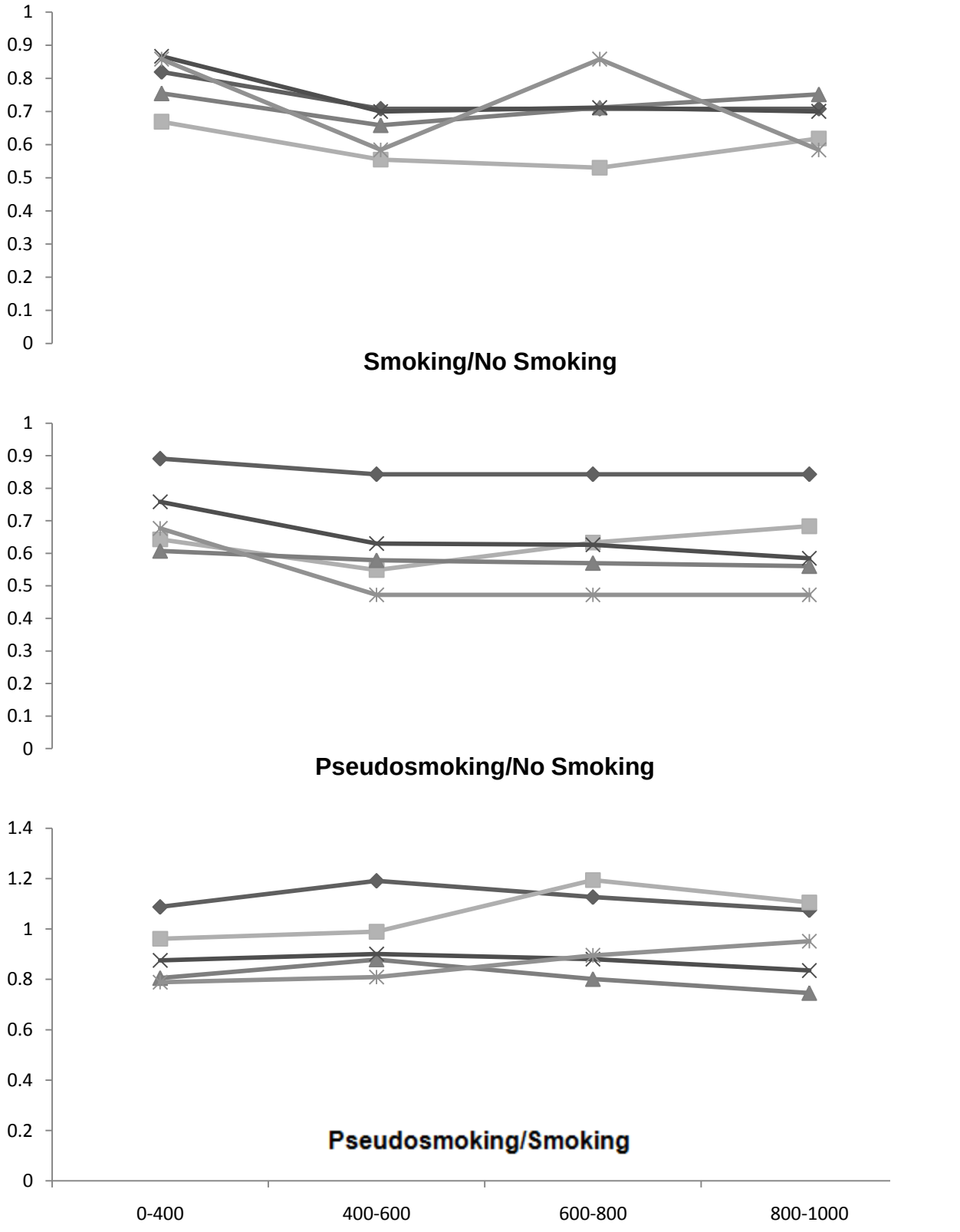

Figure 20. Capacity Ratios for XO Configuration

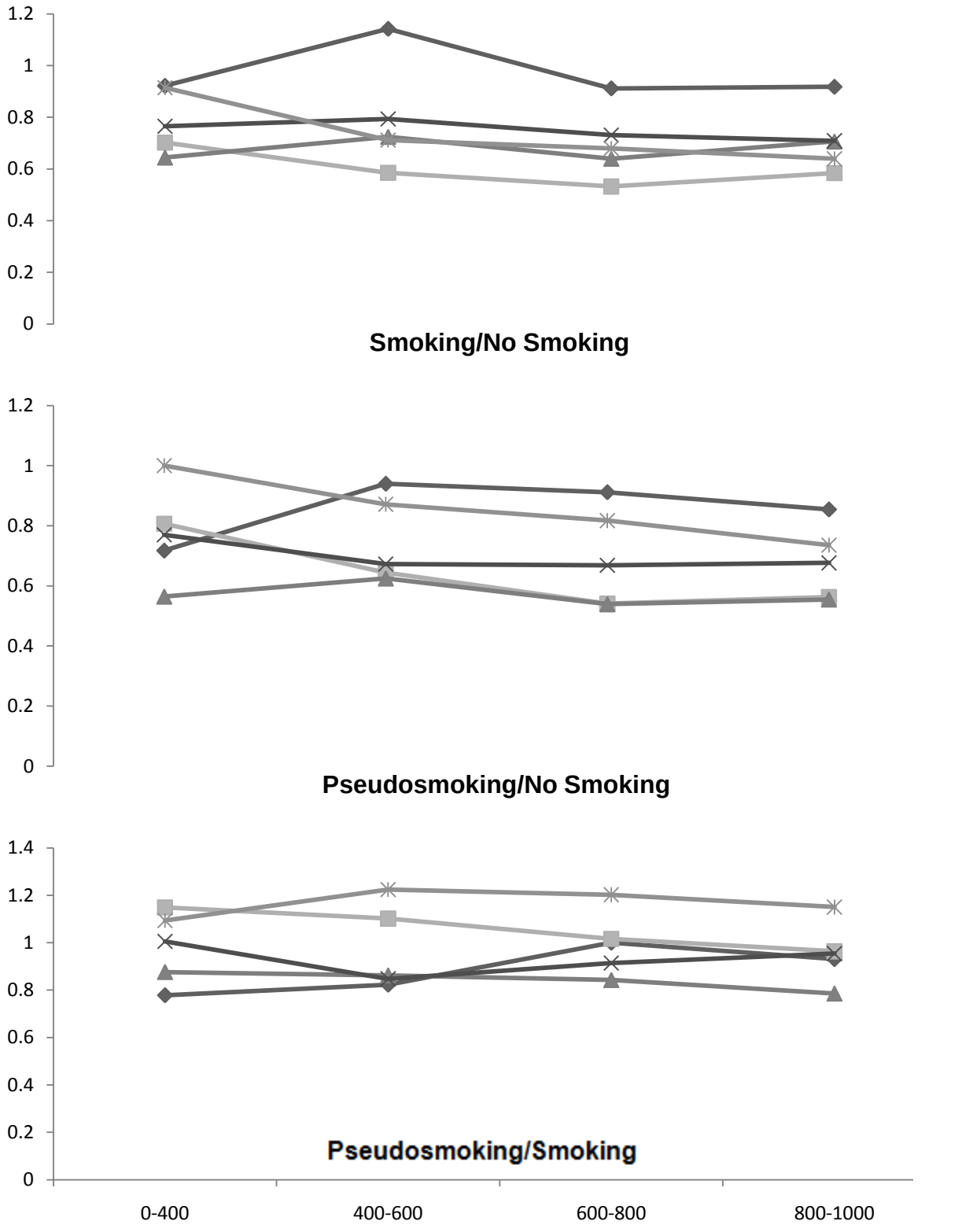

Figure 21. OX configuration

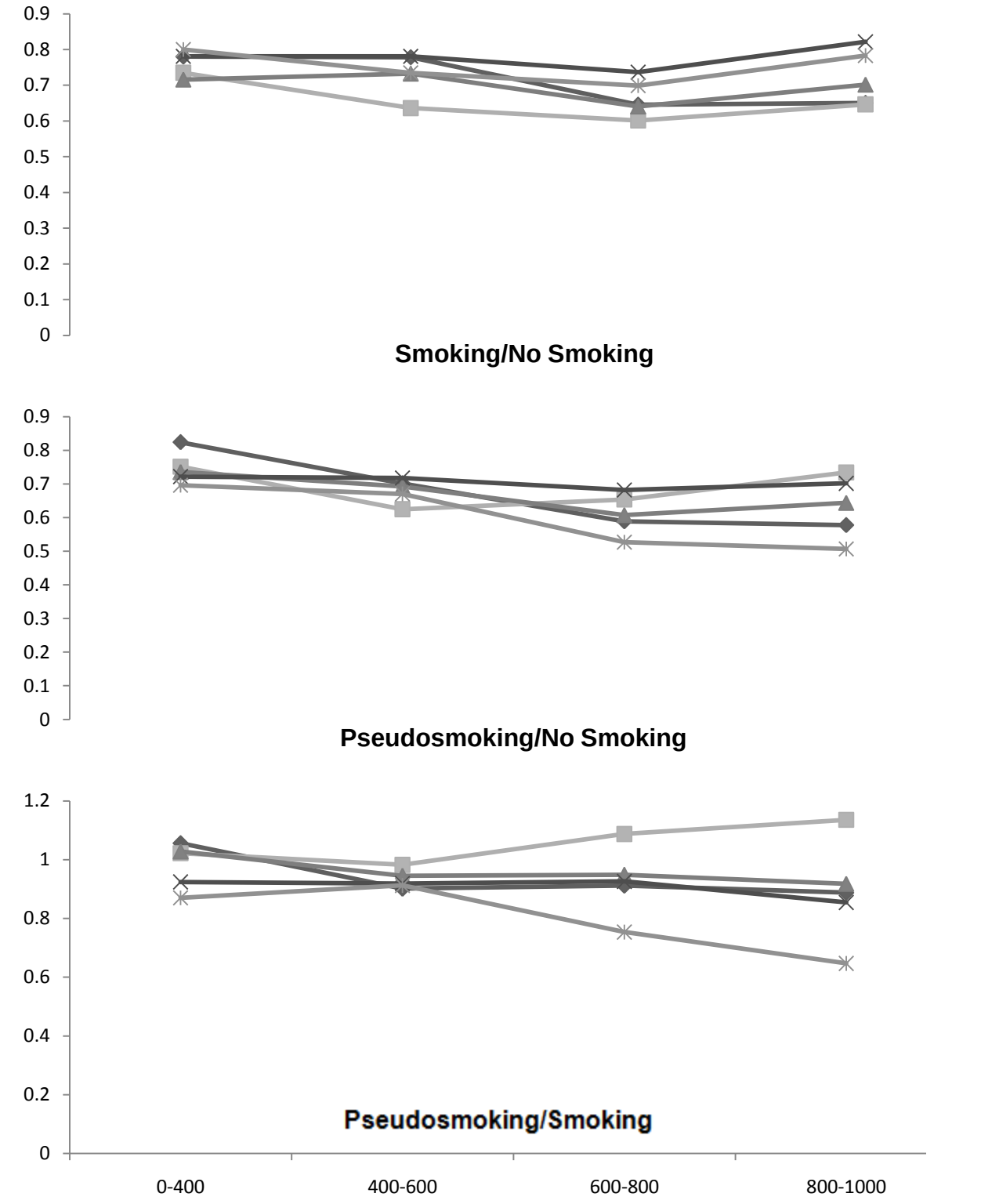

Figure 22. O_ configuration

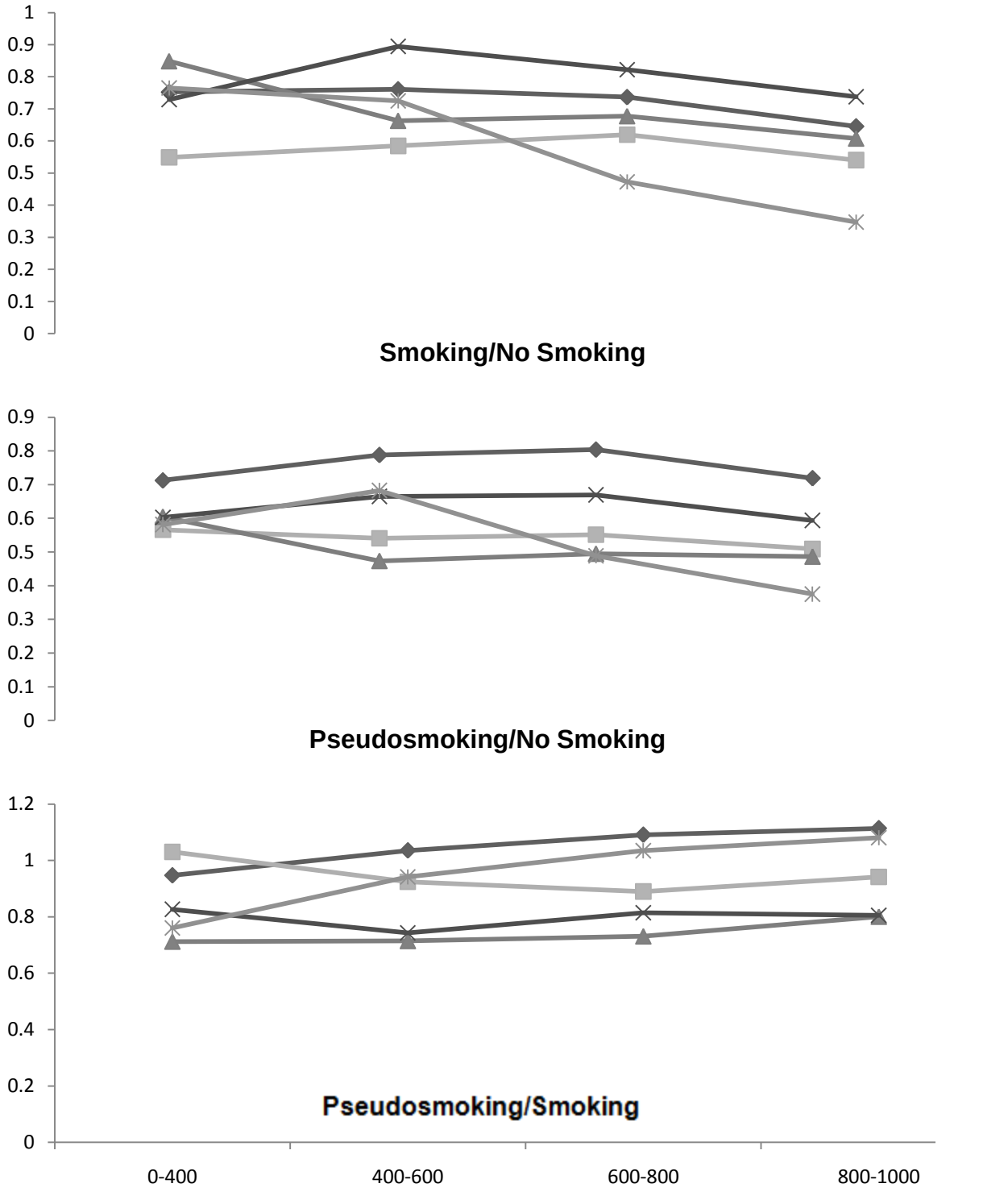

Figure 23. _O configuration

Capacity ratios comparing pseudosmoking and smoking conditions were consistently less than 1 across stimulus configurations, indicating limited capacity. The only exception was for the OX configuration, in which the moderate and daily smokers/high co-users had capacity ratios around 1 in the 0-400 ms bin, indicating unlimited capacity.

The capacity ratios comparing pseudosmoking and smoking did differ between smoking groups. Overall, the light smokers/high co-users had capacity ratios greater than 1 (particularly evident in the XX and X_ configurations), indicating that smoking taxed greater capacity than did pseudosmoking. The capacity demands of pseudosmoking were less than that of smoking. For light smokers/low co-users the results were less consistent across stimulus configurations. For some configurations, such as XX and X_, capacity ratios were less than 1. However, for other configurations, capacity ratios were greater than 1. There was no clear pattern for this group. For the moderate and daily smokers, who were low alcohol and cigarette co-users, the capacity ratios tended to be lower than 1, indicating limited capacity. Inconsistent results were found for the moderate and daily smokers/high co-users across stimulus configurations.

Discussion

The goal of the current analysis was to examine whether participants who were high co-users of alcohol and cigarettes had a different pattern of capacity ratios than those who did not co-use alcohol and cigarettes with regularity. It was very clear that light smokers within the current sample had a very high rate of alcohol and cigarette coupling. Over 70% of these smokers used cigarettes in conjunction with alcohol more than sixty percent of the time that they used cigarettes. It was hypothesized that these alcohol and cigarette high co-users may differ from light smokers who did not couple cigarettes and alcohol with such a high frequency. More specifically, it was hypothesized that the high co-users would have lower capacity ratios than the low co-users, as they were performing the reaction time test under conditions, i.e., no alcohol, that did not mimic their usual cigarette consumption. This hypothesis was supported. Light smokers who coupled cigarettes and alcohol frequently tended to have the lowest capacity ratios when

comparing smoking and no smoking conditions.

This difference in capacity ratios between the two groups of light smokers may be for a number of reasons. Light smokers who were high co-users may have been disadvantaged due to state dependent learning. If the majority of their smoking occurs under alcohol use conditions, it is reasonable to believe that their recall of smoking behaviour would be poorer due to the lack of alcohol use during the reaction time task. One way to investigate this possibility would be to replicate the study including the use of alcohol as a condition. It would be interesting to investigate whether capacity ratios do increase if light smoker/high co-users are able to drink alcohol when smoking. If state dependent learning was a factor, one would expect that the light smokers/high co-users capacity ratios under alcohol conditions would be equivalent to those of the light smokers/low co-users under no drinking conditions.

Another possible reason for the poorer performance of the light smoking/high co-users is that the presence of alcohol during the normal course of smoking has impaired the learning of smoking behaviour. Again, by employing a condition that incorporates alcohol use, it would be possible to compare capacity ratios under alcohol use and no alcohol use conditions. If capacity ratios for light smokers/high co-users remained low under alcohol conditions, then this may indicate that it is the encoding of smoking behaviour that is impacted by alcohol, and not the retrieval conditions.

These possibilities highlight the differences between encoding and retrieval of information, a key distinction in memory research. Based on Tulving's encoding specificity principle (Tulving & Thomson, 1973) the two processes appear quite similar. Items that are encoded in a specific way are best retrieved under similar circumstances. However, research using the dual-task paradigm has highlighted dramatic differences between the two processes. Baddeley, Lewis, Eldridge and Thomson (1984) demonstrated that a secondary task performed while encoding information affected later memory performance, whereas performing the same secondary task during the retrieval phase had virtually no effect. Encoding novel information appears to be an attention

intensive activity, whereas retrieval is not. It has been posited that encoding is an intensive attention-requiring process, whereas recall is automatic.

In the example of alcohol and cigarette co-use, it is possible that alcohol is a factor in both encoding and retrieval. If smoking behaviour is learned while using alcohol, this may impact the encoding of the steps required in order to smoke. As previously discussed, smoking is comprised of numerous behavioural components. The coupling of cigarettes and alcohol may impact the learning of all of these, or possibly, only some of these steps. The capacity ratios that compared pseudosmoking and smoking behaviour were mostly greater than one for light smokers who frequently couple alcohol and cigarettes together, suggesting that the inhalation of smoke was a key behavioural component that required mental processing. It is possible that alcohol does interfere with the learning of this behavioural component. This makes intuitive sense as inhaling smoke is a very novel behaviour, whereas the motor arm movements in order to bring the cigarette to and from the mouth are relatively common. People learn how to bring things to their mouths, such as food or drink, from a very young age, so that component of smoking behaviour may be less cognitively taxing.

The results of the current analysis did replicate the findings of Analysis 1 in regards to the capacity ratios comparing the pseudosmoking and no smoking conditions. Overall, the ratios were less than 1, indicating that pseudosmoking is capacity tax in comparison to no smoking. Again, it is impossible to say why capacity is limited for all groups, regardless of smoking group and alcohol and cigarette co-use.

The capacity ratios comparing pseudosmoking and smoking were inconsistent across stimulus configurations. The light smokers/high co-users tended to have capacity ratios greater than 1, indicating that pseudosmoking was less capacity taxing than smoking. This is consistent with the conclusion of Analysis 1, in that light smokers tended to show super capacity between these two conditions. Light smokers/low co-users however, were more inconsistent. For some stimulus configurations capacity ratios were greater than 1, and for other stimulus configurations, capacity ratios were less than 1. These

inconsistencies may be due to the fact that response times were only available for 5 individuals who were both light smokers and low co-users. The results may be more stable if a greater sample size was available. The inconsistency may also suggest that results are dependent on stimulus configurations, however there was no clear pattern to capacity ratios across different types of configurations (i.e. presentations including X or O). These inconsistencies may also suggest that light smokers/low co-users are an oddity. This group is composed of individuals who do not smoke on a regular basis, and their smoking is not related to drinking alcohol. Their smoking may be very different, from both a behavioural and mental processing perspective, than other groups. Of course, this is speculative and further research is needed in order to assess characteristics of this type of smoker.

Capacity ratios for the pseudosmoking and smoking conditions for moderate and daily smokers tended to be less than 1, regardless of co-use status. This replicates that results of Analysis 1, in that more experienced smokers tended to require greater capacity in order to cease the smoking behaviour. Daily smokers/low co-users tended to have the most consistent results, indicating limited capacity, but there were some anomalies in these findings. This was similar to the findings for the moderate smokers/low co-users.

The findings for the moderate and daily smokers/high co-users did not have a clear pattern of findings, with an almost equivalent mixture of capacity ratios less than, equal to, or greater than 1. As with the light smokers/low co-users, this was a relatively small group with only 6 participants included. Caution should be exercised in the interpretation of the results. The inconsistencies in results may again suggest that this group is quite different than other smokers. If state dependent learning is a factor, these smokers may perform more consistently under alcohol use conditions. If capacity ratios increased under alcohol use conditions, then this would provide evidence for state dependent learning. If capacity ratios remained the same however, this would indicate that alcohol may be interfering with learning smoking behaviour, even after a great deal of smoking experience. This would suggest that alcohol use itself is a more powerful influence on learning then experience.

There are significant limitations to the current analysis. It is unknown whether smoking behaviour was initially learned solely under high alcohol and cigarette co-use situations. Information about alcohol and cigarette co-use was only collected for the previous 30 days, and it is impossible to know whether smoking learning occurred only under alcohol use conditions. A prospective study tracking smoking and alcohol use through adolescence and young adulthood would provide a better assessment of the co-use of alcohol and cigarettes, and how this impacts capacity. If it was certain that initial smoking behavior solely took place while using alcohol, more definitive conclusions could be made about the impact of alcohol on learning smoking behavior. This would also potentially help clarify whether alcohol is impacting encoding or retrieval.

The current analysis is only a starting point in considering the relationship between alcohol use and its potential impact on the learning of smoking behaviour. It also cannot be ignored that both alcohol and cigarettes have both individual and combined physiological effects, which may impact learning of smoking behaviour. It would be simplistic to conclude that it is only the combined presence of alcohol and nicotine that may have an impact on learning. Clearly there is a great deal of further research that is required in order to clarify the relationship between alcohol use, cigarettes, and capacity ratios.

General Discussion

The goals of the current series of analyses were numerous. The main focus was to investigate whether smoking was an automatic process, using capacity as an indicator of automatic processing. By utilizing mathematical modeling, it was hoped to more clearly identify differences in the mental processing that occurred in different types of smokers under various smoking conditions. These conclusions reached on the basis of the mathematical methods were then compared to conclusions made from traditional analysis of variance, to investigate whether capacity ratios and coefficients did provide more information about performance under the different smoking conditions. Finally, capacity ratios were compared between individuals who frequently coupled alcohol and cigarettes and those who were infrequent couplers. Given alcohol's possible effects on both encoding and retrieval of information, it was posited that the mental processing of individuals who coupled alcohol and cigarettes frequently may be different in comparison to low frequency co-users.

Overall, it appears that smoking, regardless of the level of smoking experience of smokers in the current study, does make demands on cognitive processing. Results using both capacity measures and analysis of variance supported this conclusion. Capacity ratios comparing cognitive processing requirements for the smoking condition and no smoking conditions where consistently less than 1, indicating limited capacity. Results using a traditional ANOVA also indicated that reaction times were significantly faster in the no smoking condition in comparison to the smoking condition, regardless of smoking group. Whether one uses capacity as an indicator of automaticity or uses mean reaction times as a measure of automaticity the overall results do not support the idea that smoking had become automatic, even in the daily smokers.

Capacity ratios did increase with increased smoking experience. While the non-smokers capacity ratios ranged from 0.44-0.72, daily smokers' capacity ratios ranged from 0.5-0.88. This increase in capacity ratios suggest that with increased smoking experience, mental processing requirements for engaging in smoking behaviour are reduced. The

current sample had relatively low daily consumption rates and low dependence levels in comparison to other samples of smokers, so it would be worthwhile to replicate the current study with heavier smokers.

The pseudosmoking condition was also consistently, regardless of analysis type, found to be cognitively taxing for all smoking groups. When capacity ratios were calculated comparing cognitive demands for pseudosmoking compared to no smoking, the ratios were less than 1 for all groups. Similarly, reaction times were significantly slower in the pseudosmoking condition than in the no smoking condition. While one may argue that there were different reasons among groups for the reduced performances in the pseudosmoking condition, there is no direct evidence for this.

In Analysis 1, capacity ratios comparing pseudosmoking and smoking revealed differences between smoking groups. For non- and light smokers, capacity ratios were greater than one, indicating super capacity. When the demand to complete the smoking behaviour was eliminated, mental processing was improved. For moderate and daily smokers, capacity ratios were less than one, indicating limited capacity. The cessation of smoking behaviour taxed mental processing. This difference in capacity ratios seems to highlight that there may be components of smoking behaviour that are more capacity taxing then others. These results suggested that it may be prudent to utilize a feature based approach to the study of smoking behaviour.

Capacity ratios were also utilized to investigate the relationship between alcohol and cigarette co-use. Smokers are more likely to use alcohol than non-smokers, and light smokers in particular are more likely to smoke in the presence of alcohol than to smoke independently of alcohol use. This was found to be the case in the current sample. A vast majority of light smokers frequently coupled alcohol and cigarettes. Conversely, very few moderate and daily smokers were high co-users of cigarettes and alcohol.

When examining capacity ratios among high and low co-users, differences were found in the cognitive demands of the various smoking conditions. In Analysis 1, light smokers

were found to display super capacity when comparing pseudosmoking and smoking. However, in Analysis 3 when smokers were distinguished according to level of co-use of alcohol and smoking, only light smokers who were high co-users showed super capacity. Light smokers who were low co-users, displayed more inconsistent patterns of results.

These inconsistencies in capacity ratios may have been due to the small sample size of light smokers/low co-users, or an indication that this group is a unique group in their capacity demands. It would be interesting, although incredibly difficult, to capture moment to moment capacity demands of smoking. It may be that smoking impacts capacity differently depending on what component of smoking behaviour a person is engaging in. Given the overall differences between smoking groups in capacity ratios comparing pseudosmoking and smoking conditions, this indicates a difference in mental processing during various parts of the smoking behaviour. It would also be interesting to examine whether smoking of the first half of the cigarette impacts capacity differently than the second half. Light smokers especially may need some time to get accustomed to smoking a cigarette. It would be interesting to study whether mental processing changes over the course of smoking a cigarette.

Awareness of Automatic Behaviours

Despite the fact that cognitive capacity is taxed during smoking, smokers themselves describe smoking as automatic (Johnson et al., 2005). Smoking may be experienced as automatic if the cognitive resources that are required for smoking are so minimal that they are not consciously noticed by the smoker. Smoking is not perceived by the smoker as needing attention. However, as this research does indicate, cognitive capacity is required in order to smoke. This need for cognitive awareness is problematic when considering that smokers often engage in other activities while smoking.

In utilizing the capacity coefficients, it was found that different types of stimulus configurations impacted capacity in different ways. Double targets limited capacity in comparison to the sum of single targets, whereas the comparison of double non-targets to

single non-targets indicated super capacity. Despite the differences in mental processing requirements for the different stimulus configurations, the pattern of capacity ratios for smoking groups remained consistent across stimulus configurations. These results suggested that the complexity of the secondary task does not appear to impact the cognitive requirements of smoking behaviour. This is relevant, as smokers perform numerous secondary tasks while engaging in smoking behaviour. They drive cars, have conversations, drink and eat, and engage in numerous other activities while smoking. The capacity coefficient results suggest that the mental requirements of the secondary task do not modulate the mental capacity requirements for smoking behaviour. Smoking may require the same amount of processing whether someone is doing something relatively simple, or something more complex.

It is of concern that smokers engage in many types of secondary behaviours while smoking, without realizing that smoking requires some level of mental processing. Many smokers engage in complex tasks such as driving a car. While the deleterious effects of alcohol, marijuana, and other substances on driving are well established, cigarette smoking has been widely ignored. This oversight is likely due to the fact that cigarettes are not thought to be debilitating like other substances. However, the current research suggests that it is not only intoxicating effects of substances that are a concern, but also the mental processing resources that they require. Studies that examine drivers' response times under smoking conditions would be beneficial in understanding the effect smoking cigarettes has on driving.

Capacity and Smoking Groups

For the current series of studies, participants were grouped based on smoking in the last 30 days; non-smokers, light smokers, moderate smokers and daily smokers. These groups differed not only in number of days smoked in the last 30 days, but also in demographics, smoking histories, and dependence levels. This manner of classifying smokers is consistent with other research that used discrete categories of smoking such as never, former, and current (Martini, Wagner, & Anthony, 2002). Other methods of studying

smoking status include single question rating scales ranging from never smoked a cigarette to two packs a day (Repetto et al., 2005), to more detailed methods such as estimating daily number of cigarettes smoked for each year that a participant has smoked (Baxter & Hinson, 2001). While these methods can be quite useful, other methods of classifying smokers should be explored.

Defining typologies of smokers is a vital, yet underdeveloped, component of smoking research. Identifying subtypes of smoking groups may be beneficial in understanding smoking behaviour and providing treatment to stop the behaviour. Group subtypes have been found in alcoholism (Babor et al., 1992), opiate use (Zinberg & Johnson, 1976), and even gambling behaviour (Blaszczynski & Nower, 2002). These subtypes have proven to be useful in conceptualizing these behaviours, as well as treating them. For example, Type A alcoholics, defined as those with a later age of onset of alcohol problems, fewer childhood risk factors and fewer psychiatric symptoms, have shown better treatment response rates using SSRI‘s than Type B alcoholics, those with who earlier onset of alcohol problems, more childhood risk factors and more psychiatric problems (Dundon, Lynch, Pettinati, & Lipkin, 2004).

The current analyses supported a classification system that incorporates alcohol co-use: differences in capacity ratios that were not evident in Analysis 1 were found among smoking groups when alcohol co-use was considered in Analysis 3. This suggests that classifying smokers on smoking variables alone may not be adequate. There are likely numerous other variables that may be used to distinguish smokers who have the same level of smoking. However, within the current context of mental processing, it appears that alcohol co-use is a very important consideration.

In addition to a lack of a standard classification system for smokers, the assessment of the emergence of tobacco dependence has been recognized as an important, yet understudied, issue in smoking research (Tiffany, Conklin, Shiffman & Clayton, 2004). There have been some proposed stages in smoking development. These stages include contemplation, when an individual begins thinking about smoking, initiation or

experimentation with cigarettes, gradual increase in the frequency of smoking, regular smoking (less sporadic than previous), and daily/almost daily smoking as the final stage (Leventhal & Cleary, 1980). However, Mayhew, Flay & Mott (2000) found no consistent evidence for these stages in the literature. They suggest that these inconclusive findings are due to lack of standard operational definitions of these stages.

As shown by the current research, it appears that the level of cognitive processing that is required in order to smoke is related to both smoking experience and level of dependence. As the frequency of smoking behavior increased, the cognitive requirements for engaging in smoking behavior decreased. Capacity ratios did increase across smoking groups when comparing smoking and no smoking. The level of cognitive processing that is required may be a marker for where on the smoking continuum an individual sits. This is consistent with McFall and Townsend's (1989) argument that mathematical modeling could be a powerful tool in the area of psychological assessment. Capacity provides valuable information about a smoker's cognitive requirements. This information can be garnered without relying on self-report, which is how the bulk of information about one's smoking status is usually collected.

Another advantage of using capacity measures in the assessment of smoking is that it can help conceptualize substance use as a process, rather than a static state. Conceptualizations about substance use issues are often categorical and discrete. Substance use dependence and abuse, as defined by the DSM-IV-TR (2004), is categorical in nature. One must meet a number of diagnostic criteria in order to receive a diagnosis. Much of the research on addictive behaviours looks at stable factors prior to the development of substance use, such as parental substance use, history of deviant behaviour, or prior use of other substances (Chassin, Presson, Pitts & Sherman, 2000; Juon, Ensminger, & Sydnor, 2002). While important, these approaches fail to address the processes involved in the development of substance use. The use of capacity measures illustrates the process, from a cognitive perspective. With smoking experience, capacity measures change.

Treatment Implications

The current gold standard for treatment of smoking behaviour is the combination of pharmacological interventions, such as Champix or nicotine replacements, and behavioural interventions (Mojica et al., 2004). However, quit rates remain relatively low. In addition to the combination of pharmacological treatments and behavioral approaches, other quitting strategies include using pharmacological treatments on their own, quitting cold turkey, hypnosis, laser therapy, and through sheer willpower. However, many of these methods are not scientifically validated, and often have high relapse rates.

The fact that smoking does require cognitive resources is positive news in terms of treatment implications. If smoking was truly automatic, this could pose difficulties in treatment. Shiffrin & Schneider (1977) found that unlearning an automatic memory search set and learning a new search set takes longer than learning the original search set. This suggested that the original, automatic set somehow interfered with learning new material. It has also been found that there are changes in brain activity when trying to replace previously automatic information with new information (Kubler et al., 2006).

Smokers' own beliefs that their smoking is automatic may interfere with their confidence that they can quit. Substance use may be viewed as something outside of a person's control, which may decrease feelings of self-efficacy in ability to quit the substance use. However, if a patient that is trying to quit smoking is informed that smoking does require mental processing, this may contradict their beliefs that smoking is automatic. The need for capacity may be used by a treating clinician as an explanation of how mental control is required in order to smoke, and that smoking behaviour does not have a life of its own.

Capacity ratios could be used in order to assess if there are cognitive changes that occur in the quitting process. If an individual's capacity ratios comparing smoking and no smoking increased while they were quitting, this could indicate that smoking is becoming "unlearned.", or at least less automatic. The ability to show smokers who are trying to

quit that there are changes happening, maybe even when overt smoking behavior is not, may be very beneficial. However, if capacity ratios remain unchanged, this may suggest that once smoking has developed to the level of heavy use it may have relatively more persistent effects on cognitive processing.

Redefining Controlled and Automatic Processing

While the overarching purpose of the current series of studies was to determine whether smoking was an automatic process, it is debatable whether it was truly testing "automaticity." As previously described, there is no standard and widely accepted definition of automatic and controlled processing (Moors & De Houwer, 2006). While these concepts intuitively make sense, and there is no lack of description about this dual view of cognitive processing, an empirically validated definition does not exist. For the current study, capacity was used as a marker of automaticity. While capacity is one reasonable indicator of automaticity, it is not interchangeable with "automatic processing."

One of the main criticisms of the concepts of controlled and automatic processing is that there is no description of the mechanisms underlying the transition from controlled to automatic (Birnboim, 2003). While Shiffrin and Schneider (1977) outline the steps involved in the transition from controlled to automatic, these are largely descriptive and have not been directly studied. More recently, Schneider and Chein (2003) have proposed a more complex model of controlled and automatic processing, the CAP2. The CAP2 attempts to "capture the computational richness of the diverse neuronal assemblies that comprise cortical modular columns, which are found to recur throughout the cortex with regionally specialized connection patterns". While ambitious, this model has not been empirically studied or validated. It has been proposed that the automatic/controlled distinction is best used to frame an argument about basic information processing rather than to provide a detailed description of the processes required in any individual task or set of tasks (Birnboim, 2003).

One method of examining the transition from controlled to automatic processing would be through the use of capacity measures. Capacity ratios could be used as a baseline measure prior to the learning of a task. As the learning of the task occurs, measuring capacity ratios repeatedly could provide information as to how the capacity requirements change with practice. If a new task is initially capacity limiting, a switch to unlimited capacity would indicate that a significant level of learning has taken place. This tracking of capacity measures would provide empirical evidence for a transition from a capacity limiting process to a more "automatic" one.

Mathematical Modeling in Clinical Science

The current status of the use of mathematical modeling in clinical science is limited at best. Reasons for this lack of use are undoubtedly numerous. One reason would likely be limited opportunities for receiving training in psychology research programs. A lack of awareness about these methods also influences the lack of use in mathematical modeling. McFall and Townsend (1998) argue that an integration of clinical and cognitive sciences would lead to significant advance in psychological assessment. One of the main goals of this series of studies was to provide a clinically relevant example of using these mathematical tools.

Another possible reason for the limited use of mathematical models in clinical science is that it has been difficult to conceptualize how one would actually use mathematical modeling. Wading through concepts such as architecture, capacity, and stopping rules makes the application of mathematical modeling seem quite limited and specific to memory search tasks. While the current series of studies used a traditional search and response task, it provides clinically useful information.

Another example of an application of mathematical modeling, with clinical relevance, was a recent study by Johnson, Blaha, Houpt, & Townsend (2010). This study looked at mental processing within a sample of participants diagnosed with autism spectrum

disorder (ASD), and a control sample. Overall, they found that fundamental differences in mental processing between the two groups, particularly in regards to stopping rules and dependency. This study highlights the ability to use mathematical modeling in order to highlight both strengths and weaknesses among different clinical groups. In his discussion of the Bull-in-a-Worcester-China-Shop syndrome, Maher (1974) argues that clinically disturbed populations do poorly on most measures of cognitive ability. Evidence that there are deficits in these clinical populations is not difficult to obtain. However, this may not necessarily be the most useful information. What is equally, if not more interesting, is what processes are functioning at normal levels. By obtaining this information, clinicians would be able to adapt treatment to take advantage of those functioning efficiencies. However, it is only by using mathematical applications that these processes are able to be teased apart.

Future Research

The current series of studies has provided evidence that smoking does tax capacity, but capacity demands decrease as smoking experience and dependence levels increase. While capacity was used as a marker for automaticity, other components of information processing models were not considered. These components include architecture, stopping rules and independence. Designing experiments that are able to access additional mental processing components may provide additional information about the cognitive requirements of smoking behaviour. These other mental processes could be utilized in order to study smoking as a more feature based approach.

As previously discussed, automatic processing can be considered in relation to the UCIP model (Townsend & Wenger, 2004). The current study has addressed the issue of capacity, from a pure insertion perspective. When smoking behaviour is inserted, it does affect the processing of the secondary task. The processing of the secondary task does not fit the UCIP model, as capacity was not unlimited when smoking was inserted, regardless of smoking group membership. However, it remains unknown how the insertion of smoking behaviour affects the independence and architecture of the

processing of the secondary task. System's Factorial Technology, and its associated Double Factorial Design, (Townsend & Ashby, 1983; Townsend & Nozawa, 1995) is an experimental approach that allows for the assessment of all components of processing within a single experimental design. Johnson et al. (2010) implemented such a design in order to assess all components of information processing models within their ASD sample.

In terms of general processing, it would be interesting to study how capacity requirements are perceived by individuals. While smoking does require cognitive capacity, smokers do describe their smoking as automatic. While not directly studied, many of the non- and light smokers commented during the task that they felt that smoking was very distracting, and it was difficult to do the monitoring task. It would be interesting to more formally study individuals' perceptions of effort and their corresponding capacity requirements. There may be a certain capacity cut-off that feels "automatic" or "controlled."

One critical question that may be explored through the use of capacity measures is whether there is any single behaviour that is truly "automatic." Many behaviours feel as if they require no effort or conscious awareness; however it is unknown whether there is any behaviour that truly requires no cognitive effort. Even physiologically automatic behaviours, such as breathing, may take a level of cognitive processing that is not understood. Capacity measures, as well as other components of information processing systems, could be utilized in order to explore the cognitive processing requirements of numerous, seemingly "automatic" behaviours.

In conclusion, the application of capacity measures did add significantly to the understanding of the cognitive processes underlying smoking behaviour. Its use in highlighting the importance of studying different components of smoking behaviour cannot be minimized. These capacity measures also provided solid evidence that the relationship between cigarettes and alcohol coupling also impacts cognitive processing. The use of these measures may be invaluable in better understanding substance use, and

should be utilized in future research.

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Appendix A.

UWO ETHICS NUMBER	12702S
LOCAL PRINCIPAL INVESTIGATOR	Dr. Riley Hinson
PROJECT TITLE	Smoking as an Automatic Process: Using Capacity Coefficients Among Distinct Typologies of Smokers.
Signature of Principal Investigator:	Date: April 30, 2007

1.	Review type required? (Number of copies)	FULL BOARD (Original + 16 copies)	
		EXPEDITED (Original only)	X
		FYI only TO REB, APPROVAL NOT REQUIRED (Original only)	

2.	Do the proposed changes alter the information contained in the UWO protocol submission, Letters of Information and Consent documentation or affect local participants?	YES	X
		NO	

3.	Have you included an updated company protocol or investigator's brochure with this request? (*Do not submit duplicates of information already submitted to the HSREB*.)	YES	
		NO	X
3a.	If YES, what is the date or reference number on these documents?		

4.	Does this revision require Health Canada approval? (This section is for clinical drug trials only.) **If YES, attach a copy of the No Objection letter (NOL) from Health Canada that relates to this revision. N.B. UWO HSREB approval will not be granted until a copy of the No Objection letter is received.**	YES – NOL attached	
		YES – NOL pending	
		NO	
		NA - Not a Clinical Trial	X

5	**SUMMARY OF CHANGES IN THIS REQUEST FOR A REVISION**	**√ IF YES**	**IF YES TO ANY ITEM IN THIS CHART, PROVIDE ADDITIONAL INFORMATION ON A SEPARATE SHEET AND/OR DOCUMENTATION AS NOTED BELOW. (Put Ethics # on each additional page)**
Study end date?		√	Provide revised date and detailed explanation/rationale for change.

Appendix B.

Table 1.

Observed Bin Proportions, Estimated Survivor Functions and Integrated Hazard Functions for Non-Smokers In Non-Smoking Condition

	0-400 ms	400-600 ms	600-800 ms	800-1000 ms
XX				
Observed proportion	0.908	0.070	0.015	0
Estimated survivor function	0.092	0.022	0.007	0.007
Integrated Hazard Function	2.386	3.817	4.962	4.962
X _				
Observed proportion	0.898	0.068	0.023	0.005
Estimated survivor function	0.102	0.034	0.011	0.006
Integrated Hazard Function	2.283	3.381	4.510	5.116
_ X				
Observed proportion	0.773	0.17	0.035	0.008
Estimated survivor function	0.227	0.057	0.022	0.014
Integrated Hazard Function	1.483	2.865	3.817	4.269
XO				
Observed proportion	0.810	0.115	0.058	0.013
Estimated survivor function	0.190	0.075	0.017	0.004
Integrated Hazard Function	1.661	2.590	4.074	5.521

	0-400 ms	400-600 ms	600-800ms	800-1000ms
OX				
Observed proportion	0.625	0.26	0.055	0.035
Estimated survivor function	0.375	0.115	0.06	0.025
Integrated Hazard Function	0.981	2.163	2.813	3.689
OO				
Observed Proportion	0.815	0.14	0.018	0.013
Estimated Survivor function	0.185	0.045	0.027	0.014
Integrated Hazard Function	1.687	3.101	3.612	4.269
O_				
Observed proportion	0.75	0.183	0.033	0.02
Estimated survivor function	0.25	0.067	0.034	0.014
Integrated Hazard Function	1.386	2.703	3.381	4.269
_O				
Observed proportion	0.768	0.148	0.045	0.02
Estimated survivor function	0.232	0.084	0.039	0.019
Integrated Hazard Function	1.461	2.477	3.244	3.963

Table 2.

Observed Bin Proportions, Estimated Survivor Functions and Integrated Hazard Functions for Non-Smokers in Smoking Condition

	0-400 ms	400-600 ms	600-800 ms	800-1000 ms
XX				
Observed proportion	0.783	0.093	0.033	0.018
Estimated survivor function	0.217	0.124	0.091	0.073
Integrated Hazard Function	1.528	2.087	2.397	2.617
X _				
Observed proportion	0.698	0.13	0.045	0.023
Estimated survivor function	0.302	0.172	0.127	0.104
Integrated Hazard Function	1.197	1.760	2.0636	2.263
_ X				
Observed proportion	0.633	0.183	0.055	0.033
Estimated survivor function	0.367	0.184	0.129	0.096
Integrated Hazard Function	1.002	1.693	2.048	2.343
XO				
Observed proportion	0.673	0.135	0.085	0.038
Estimated survivor function	0.327	0.192	0.107	0.069
Integrated Hazard Function	1.118	1.650	2.235	2.674

	0-400 ms	400-600 ms	600-800 ms	800-1000 ms
OX				
Observed proportion	0.51	0.235	0.093	0.033
Estimated survivor function	0.49	0.255	0.162	0.129
Integrated Hazard Function	0.713	1.366	1.820	2.048
OO				
Observed proportion	0.655	0.15	0.075	0.018
Estimated survivor function	0.345	0.195	0.12	0.102
Integrated hazard function	1.0642	1.635	2.120	2.283
O_				
Observed proportion	0.558	0.238	0.07	0.028
Estimated survivor function	0.442	0.204	0.134	0.106
Integrated Hazard Function	0.816	1.590	2.010	2.244
_O				
Observed proportion	0.585	0.23	0.053	0.045
Estimated survivor function	0.415	0.185	0.132	0.087
Integrated Hazard Function	0.879	1.687	2.025	2.442

Table 3.

Observed Bin Proportions, Estimated Survivor Functions and Integrated Hazard Functions for Non-Smokers in Pseudo-Smoking Condition

	0-400 ms	400-600 ms	600-800 ms	800-1000 ms
XX				
Observed proportion	0.745	0.123	0.028	0.033
Estimated survivor function	0.255	0.132	0.104	0.071
Integrated Hazard Function	1.366	2.025	2.263	2.645
X _				
Observed proportion	0.733	0.095	0.053	0.03
Estimated survivor function	0.267	0.172	0.119	0.089
Integrated Hazard Function	1.320	1.760	2.129	2.419
_ X				
Observed proportion	0.698	0.158	0.045	0.02
Estimated survivor function	0.302	0.144	0.099	0.079
Integrated Hazard Function	1.197	1.938	2.313	2.538
XO				
Observed proportion	0.713	0.13	0.058	0.028
Estimated survivor function	0.287	0.157	0.099	0.071
Integrated Hazard Function	1.248	1.852	2.313	2.645

	0-400 ms	400-600 ms	600-800 ms	800-1000 ms
XO				
Observed proportion	0.573	0.218	0.085	0.04
Estimated survivor function	0.427	0.209	0.124	0.084
Integrated Hazard Function	0.851	1.565	2.087	2.477
OO				
Observed proportion	0.615	0.205	0.053	0.065
Estimated survivor function	0.385	0.18	0.127	0.062
Integrated Hazard Function	0.955	1.715	2.066	2.781
O _				
Observed proportion	0.565	0.223	0.08	0.04
Estimated survivor function	0.435	0.212	0.132	0.092
Integrated Hazard Function	0.832	1.5512	2.025	2.386
_O				
Observed proportion	0.59	0.225	0.063	0.04
Estimated survivor function	0.41	0.185	0.122	0.082
Integrated Hazard Function	0.892	1.687	2.103	2.501

Table 4.

Observed Bin Proportions, Estimated Survivor Functions and Integrated Hazard Functions for Light Smokers in Non-Smoking Condition

	0-400 ms	400-600 ms	600-800 ms	800-1000 ms
XX				
Observed proportion	0.928	0.06	0.006	0
Estimated survivor function	0.072	0.012	0.006	0.006
Integrated Hazard Function	2.631	4.423	5.116	5.116
X _				
Observed proportion	0.912	0.072	0.008	0.004
Estimated survivor function	0.088	0.016	0.008	0.004
Integrated Hazard Function	2.430	4.135	4.828	5.521
_X				
Observed proportion	0.826	0.15	0.01	0.006
Estimated survivor function	0.174	0.024	0.014	0.008
Integrated Hazard Function	1.749	3.730	4.269	4.828
XO				
Observed proportion	0.844	0.124	0.02	0.002
Estimated survivor function	0.156	0.032	0.012	0.01
Integrated Hazard Function	1.858	3.442	4.423	4.605

	0-400 ms	400-600 ms	600-800 ms	800-1000 ms
OX				
Observed proportion	0.8	0.162	0.022	0.006
Estimated survivor function	0.2	0.038	0.016	0.01
Integrated Hazard Function	1.609	3.270	4.135	4.605
OO				
Observed proportion	0.757	0.178	0.034	0.014
Estimated survivor function	0.243	0.065	0.031	0.017
Integrated Hazard Function	1.415	2.733	3.474	4.075
O _				
Observed proportion	0.76	0.19	0.03	0.006
Estimated survivor function	0.24	0.05	0.02	0.014
Integrated Hazard Function	1.427	2.996	3.912	4.269
_O				
Observed proportion	0.838	0.126	0.016	0.012
Estimated survivor function	0.162	0.036	0.02	0.008
Integrated Hazard Function	1.820	3.324	3.912	4.828

Table 5.

Observed Bin Proportions, Estimated Survivor Functions and Integrated Hazard Functions for Light Smokers In Smoking Condition

	0-400 ms	400-600 ms	600-800 ms	800-1000 ms
XX				
Observed proportion	0.72	0.152	0.034	0.02
Estimated survivor function	0.28	0.128	0.094	0.074
Integrated Hazard Function	1.273	2.056	2.364	2.604
X _				
Observed proportion	0.722	0.144	0.028	0.018
Estimated survivor function	0.278	0.134	0.106	0.088
Integrated Hazard Function	1.280	2.010	2.244	2.430
_X				
Observed proportion	0.67	0.184	0.042	0.022
Estimated survivor function	0.33	0.146	0.104	0.082
Integrated Hazard Function	1.109	1.924	2.263	2.501
XO				
Observed proportion	0.72	0.14	0.034	0.036
Estimated survivor function	0.28	0.14	0.106	0.07
Integrated Hazard Function	1.273	1.966	2.244	2.659

	0-400 ms	400-600 ms	600-800 ms	800-1000 ms
OX				
Observed proportion	0.684	0.200	0.028	0.022
Estimated survivor function	0.316	0.116	0.088	0.066
Integrated Hazard Function	1.152	2.154	2.430	2.718
OO				
Observed proportion	0.655	0.217	0.05	0.014
Estimated survivor function	0.345	0.128	0.078	0.064
Integrated Hazard Function	1.064	2.056	2.551	2.749
O _				
Observed proportion	0.608	0.222	0.046	0.04
Estimated survivor function	0.392	0.17	0.124	0.084
Integrated Hazard Function	0.936	1.772	2.087	2.477
_O				
Observed proportion	0.654	0.206	0.056	0.014
Estimated survivor function	0.346	0.14	0.084	0.07
Integrated Hazard Function	1.061	1.966	2.477	2.659

Table 6.

Observed Bin Proportions, Estimated Survivor Functions and Integrated Hazard Functions for Light Smokers In Pseudo - Smoking Condition

	0-400 ms	400-600 ms	600-800 ms	800-1000 ms
XX				
Observed proportion	0.754	0.124	0.042	0.022
Estimated survivor function	0.246	0.122	0.08	0.058
Integrated Hazard Function	1.402	2.104	2.526	2.847
X _				
Observed proportion	0.736	0.12	0.044	0.042
Estimated survivor function	0.264	0.144	0.1	0.058
Integrated Hazard Function	0.909	1.732	2.207	2.513
_X				
Observed proportion	0.702	0.168	0.046	0.022
Estimated survivor function	0.298	0.13	0.084	0.062
Integrated Hazard Function	1.211	2.040	2.477	2.781
XO				
Observed proportion	0.714	0.16	0.056	0.02
Estimated survivor function	0.286	0.126	0.07	0.05
Integrated Hazard Function	1.252	2.071	2.659	2.996

	0-400 ms	400-600 ms	600-800 ms	800-1000 ms
OX				
Observed proportion	0.668	0.232	0.042	0.022
Estimated survivor function	0.332	0.100	0.058	0.036
Integrated Hazard Function	1.103	2.303	2.847	3.324
OO				
Observed proportion	0.624	0.202	0.057	0.034
Estimated survivor function	0.376	0.174	0.117	0.083
Integrated Hazard Function	0.978	1.749	2.146	2.489
O_				
Observed proportion	0.646	0.19	0.074	0.03
Estimated survivor function	0.354	0.164	0.090	0.06
Integrated Hazard Function	1.038	1.808	2.408	2.813
_O				
Observed proportion	0.664	0.184	0.054	0.024
Estimated survivor function	0.336	0.152	0.098	0.074
Integrated Hazard Function	1.091	1.884	2.323	2.604

Table 7.

Observed Bin Proportions, Estimated Survivor Functions and Integrated Hazard Functions for Moderate Smokers In Non-Smoking Condition

	0-400 ms	400-600 ms	600-800 ms	800-1000 ms
XX				
Observed proportion	0.883	0.083	0.014	0.009
Estimated survivor function	0.117	0.034	0.02	0.011
Integrated Hazard Function	2.146	3.381	3.912	4.510
X _				
Observed proportion	0.903	0.067	0.01	0.007
Estimated survivor function	0.097	0.03	0.02	0.013
Integrated Hazard Function	2.333	3.507	3.912	4.343
_X				
Observed proportion	0.802	0.141	0.026	0.016
Estimated survivor function	0.198	0.057	0.031	0.015
Integrated Hazard Function	1.619	2.865	3.474	4.200
XO				
Observed proportion	0.803	0.147	0.028	0.009
Estimated survivor function	0.197	0.05	0.022	0.013
Integrated Hazard Function	1.625	3.00	3.817	4.343

	0-400 ms	400-600 ms	600-800 ms	800-1000 ms
OX				
Observed proportion	0.726	0.176	0.06	0.014
Estimated survivor function	0.274	0.098	0.038	0.024
Integrated Hazard Function	1.295	2.323	3.270	3.730
OO				
Observed proportion	0.757	0.178	0.034	0.014
Estimated survivor function	0.243	0.065	0.031	0.017
Integrated Hazard Function	1.415	2.733	3.474	4.074
O _				
Observed proportion	0.7	0.222	0.053	0.01
Estimated survivor function	0.3	0.078	0.025	0.015
Integrated Hazard Function	1.204	2.551	3.689	4.200
_O				
Observed proportion	0.781	0.162	0.036	0.01
Estimated survivor function	0.219	0.057	0.021	0.011
Integrated Hazard Function	1.519	2.865	3.863	4.510

Table 8.

Observed Bin Proportions, Estimated Survivor Functions and Integrated Hazard Functions for Moderate Smokers In Smoking Condition

	0-400 ms	400-600 ms	600-800 ms	800-1000 ms
XX				
Observed proportion	0.793	0.103	0.034	0.026
Estimated survivor function	0.207	0.104	0.07	0.044
Integrated Hazard Function	1.575	2.263	2.660	3.124
X _				
Observed proportion	0.74	0.141	0.05	0.014
Estimated survivor function	0.26	0.119	0.069	0.055
Integrated Hazard Function	1.347	2.129	2.674	2.900
_ X				
Observed proportion	0.693	0.181	0.053	0.017
Estimated survivor function	0.307	0.126	0.073	0.056
Integrated Hazard Function	1.181	2.071	2.617	2.882
XO				
Observed proportion	0.71	0.145	0.06	0.033
Estimated survivor function	0.29	0.145	0.085	0.052
Integrated Hazard Function	1.238	1.931	2.465	2.957

	0-400 ms	400-600 ms	600-800 ms	800-1000 ms
OX				
Observed proportion	0.586	0.224	0.079	0.052
Estimated survivor function	0.414	0.19	0.111	0.059
Integrated Hazard Function	0.882	1.661	2.198	2.830
OO				
Observed proportion	0.655	0.217	0.05	0.014
Estimated survivor function	0.345	0.128	0.078	0.064
Integrated Hazard Function	1.064	2.0565	2.551	2.749
O_				
Observed proportion	0.619	0.25	0.067	0.021
Estimated survivor function	0.381	0.131	0.064	0.043
Integrated Hazard Function	0.965	2.033	2.749	3.147
_O				
Observed proportion	0.7	0.171	0.053	0.017
Estimated survivor function	0.3	0.129	0.076	0.059
Integrated Hazard Function	1.204	2.048	2.577	2.830

Table 9.

Observed Bin Proportions, Estimated Survivor Functions and Integrated Hazard Functions for Moderate Smokers In Pseudo - Smoking Condition

	0-400 ms	400-600 ms	600-800 ms	800-1000 ms
XX				
Observed proportion	0.743	0.148	0.029	0.019
Estimated survivor function	0.257	0.109	0.08	0.061
Integrated Hazard Function	1.359	2.216	2.526	2.797
X _				
Observed proportion	0.736	0.103	0.045	0.031
Estimated survivor function	0.264	0.161	0.116	0.085
Integrated Hazard Function	1.332	1.826	2.154	2.465
_ X				
Observed proportion	0.667	0.179	0.043	0.033
Estimated survivor function	0.333	0.154	0.111	0.078
Integrated Hazard Function	1.100	1.871	2.198	2.551
XO				
Observed proportion	0.652	0.162	0.066	0.04
Estimated survivor function	0.348	0.186	0.12	0.08
Integrated Hazard Function	1.056	1.682	2.120	2.526

	0-400 ms	400-600 ms	600-800 ms	800-1000 ms
OX				
Observed proportion	0.559	0.24	0.071	0.041
Estimated survivor function	0.441	0.201	0.13	0.089
Integrated Hazard Function	0.819	1.604	2.040	2.419
OO				
Observed proportion	0.624	0.202	0.057	0.034
Estimated survivor function	0.376	0.174	0.117	0.083
Integrated Hazard Function	0.978	1.749	2.146	2.489
O_				
Observed proportion	0.597	0.226	0.067	0.029
Estimated survivor function	0.403	0.177	0.11	0.081
Integrated Hazard Function	0.909	1.732	2.207	2.513
_O				
Observed proportion	0.591	0.212	0.072	0.036
Estimated survivor function	0.409	0.197	0.125	0.089
Integrated Hazard Function	0.894	1.6245	2.079	2.419

Table 10.

Observed Bin Proportions, Estimated Survivor Functions and Integrated Hazard Functions for Smokers In Non -Smoking Condition

	0-400 ms	400-600 ms	600-800 ms	800-1000 ms
XX				
Observed proportion	0.906	0.085	0.004	0
Estimated survivor function	0.094	0.009	0.005	0.005
Integrated Hazard Function	2.364	4.710	5.298	5.298
X _				
Observed proportion	0.923	0.052	0.013	0.006
Estimated survivor function	0.077	0.025	0.012	0.006
Integrated Hazard Function	2.564	3.689	4.423	5.116
_ X				
Observed proportion	0.835	0.142	0.012	0.008
Estimated survivor function	0.165	0.023	0.011	0.003
Integrated Hazard Function	1.802	3.772	4.510	5.809
XO				
Observed proportion	0.815	0.158	0.017	0.006
Estimated survivor function	0.185	0.027	0.01	0.004
Integrated Hazard Function	1.687	3.612	4.605	5.521

	0-400 ms	400-600 ms	600-800 ms	800-1000 ms
OX				
Observed proportion	0.681	0.24	0.052	0.015
Estimated survivor function	0.319	0.079	0.027	0.012
Integrated Hazard Function	1.143	2.538	3.612	4.423
OO				
Observed proportion	0.823	0.146	0.021	0
Estimated survivor function	0.177	0.031	0.01	0.01
Integrated Hazard Function	1.732	3.474	4.605	4.605
O_				
Observed proportion	0.75	0.194	0.04	0.004
Estimated survivor function	0.25	0.056	0.016	0.012
Integrated Hazard Function	1.386	2.882	4.135	4.423
_O				
Observed proportion	0.84	0.117	0.023	0.012
Estimated survivor function	0.16	0.043	0.02	0.008
Integrated Hazard Function	1.833	3.147	3.912	4.828

Table 11.

Observed Bin Proportions, Estimated Survivor Functions and Integrated Hazard Functions for Daily Smokers In Smoking Condition

	0-400 ms	400-600 ms	600-800 ms	800-1000 ms
XX				
Observed proportion	0.838	0.1	0.033	0.008
Estimated survivor function	0.162	0.062	0.029	0.021
Integrated Hazard Function	1.820	2.781	3.540	3.863
X _				
Observed proportion	0.835	0.117	0.019	0.01
Estimated survivor function	0.165	0.048	0.029	0.019
Integrated Hazard Function	1.802	3.037	3.540	3.963
_ X				
Observed proportion	0.74	0.167	0.038	0.029
Estimated survivor function	0.26	0.093	0.055	0.026
Integrated Hazard Function	1.347	2.375	2.900	3.650
XO				
Observed proportion	0.773	0.146	0.04	0.013
Estimated survivor function	0.227	0.081	0.041	0.028
Integrated Hazard Function	1.483	2.513	3.194	3.576

	0-400 ms	400-600 ms	600-800 ms	800-1000 ms
OX				
Observed proportion	0.579	0.277	0.071	0.029
Estimated survivor function	0.421	0.144	0.073	0.044
Integrated Hazard Function	0.865	1.938	2.617	3.124
OO				
Observed proportion	0.735	0.188	0.031	0.017
Estimated survivor function	0.265	0.077	0.046	0.029
Integrated Hazard Function	1.328	2.564	3.079	3.540
O_				
Observed proportion	0.669	0.229	0.052	0.021
Estimated survivor function	0.331	0.102	0.05	0.029
Integrated Hazard Function	1.106	2.283	2.996	3.540
_O				
Observed proportion	0.733	0.187	0.037	0.013
Estimated survivor function	0.267	0.08	0.043	0.03
Integrated Hazard Function	1.321	2.526	3.147	3.507

Table 12.

Observed Bin Proportions, Estimated Survivor Functions and Integrated Hazard Functions for Daily Smokers In Pseudosmoking Condition

	0-400 ms	400-600 ms	600-800 ms	800-1000 ms
XX				
Observed proportion	0.756	0.152	0.031	0.012
Estimated survivor function	0.244	0.092	0.061	0.049
Integrated Hazard Function	1.411	2.386	2.797	3.016
X _				
Observed proportion	0.733	0.138	0.054	0.019
Estimated survivor function	0.267	0.129	0.075	0.056
Integrated Hazard Function	1.321	2.048	2.590	2.882
_ X				
Observed proportion	0.667	0.225	0.058	0.027
Estimated survivor function	0.333	0.108	0.050	0.023
Integrated Hazard Function	1.100	2.226	2.996	3.772
XO				
Observed proportion	0.712	0.173	0.052	0.013
Estimated survivor function	0.288	0.115	0.063	0.05
Integrated Hazard Function	1.245	2.163	2.765	2.996

	0-400 ms	400-600 ms	600-800 ms	800-1000 ms
OX				
Observed proportion	0.55	0.252	0.094	0.042
Estimated survivor function	0.45	0.198	0.104	0.062
Integrated Hazard Function	0.799	1.619	2.263	2.781
OO				
Observed proportion	0.681	0.212	0.04	0.025
Estimated survivor function	0.319	0.107	0.067	0.042
Integrated Hazard Function	1.143	2.235	2.703	3.170
O_				
Observed proportion	0.621	0.248	0.06	0.023
Estimated survivor function	0.379	0.131	0.071	0.048
Integrated Hazard Function	0.970	2.033	2.645	3.0367
_O				
Observed proportion	0.662	0.204	0.048	0.033
Estimated survivor function	0.338	0.134	0.086	0.053
Integrated Hazard Function	1.085	2.010	2.453	2.937

Table 13.

Observed Bin Proportions, Estimated Survivor Functions and Integrated Hazard Functions for Complete Sample In Non-Smoking Condition

	0-400 ms	400-600 ms	600-800 ms	800-1000 ms
XX				
Observed proportion	0.905	0.075	0.01	0.003
Estimated survivor function	0.095	0.02	0.01	0.007
Integrated Hazard Function	2.354	3.912	4.605	4.962
X _				
Observed proportion	0.91	0.065	0.013	0.006
Estimated survivor function	0.09	0.025	0.012	0.006
Integrated Hazard Function	2.408	3.689	4.422	5.116
_X				
Observed proportion	0.811	0.15	0.02	0.01
Estimated survivor function	0.189	0.039	0.019	0.009
Integrated Hazard Function	1.666	3.244	3.963	4.711

	0-400 ms	400-600 ms	600-800 ms	800-1000 ms
XO				
Observed proportion	0.818	0.138	0.029	0.007
Estimated survivor function	0.182	0.044	0.015	0.008
Integrated Hazard Function	1.704	3.124	4.200	4.828
OX				
Observed proportion	0.679	0.225	0.059	0.02
Estimated survivor function	0.321	0.096	0.037	0.017
Integrated Hazard Function	1.136	2.343	3.297	4.075

Table 14.

Observed Bin Proportions, F(t) and K(t) for Complete Sample In Non-Smoking Condition

	0-400 ms	400-600 ms	600-800 ms	800-1000 ms
OO				
Observed proportion	0.797	0.158	0.025	0.008
F(t)	0.797	0.955	0.98	0.988
K(t)	-0.227	-0.0460	-0.020	-0.012
O _				
Observed proportion	0.739	0.199	0.04	0.01
F(t)	0.739	0.938	0.978	0.988
K(t)	-0.302	-0.064	-0.022	-0.012
_O				
Observed proportion	0.808	0.139	0.03	0.013
F(t)	0.808	0.947	0.977	0.99
K(t)	-0.213	-0.054	-0.023	-0.010

Table 15.

Observed Bin Proportions, F(t) and K(t) for Non-Smokers In Smoking Condition

	0-400 ms	400-600 ms	600-800 ms	800-1000 ms
OO				
Observed proportion	0.655	0.15	0.075	0.018
F(t)	0.655	0.805	0.88	0.898
K(t)	-0.423	-0.217	-0.128	-0.108
O _				
Observed proportion	0.558	0.238	0.07	0.028
F(t)	0.558	0.796	0.866	0.894
K(t)	-0.583	-0.228	-0.144	-0.112
_O				
Observed proportion	0.585	0.230	0.053	0.045
F(t)	0.585	0.815	0.868	0.913
K(t)	-0.536	-0.204	-0.142	-0.091

Table 16.

Observed Bin Proportions, F(t) and K(t) for Light Smokers In Smoking Condition

	0-400 ms	400-600 ms	600-800 ms	800-1000 ms
OO				
Observed proportion	0.684	0.200	0.028	0.022
F(t)	0.684	0.884	0.912	0.934
K(t)	-0.380	-0.123	-0.092	-0.068
O _				
Observed proportion	0.608	0.222	0.046	0.040
F(t)	0.608	0.830	0.876	0.916
K(t)	-0.498	-0.186	-0.132	-0.088
_O				
Observed proportion	0.654	0.206	0.056	0.014
F(t)	0.654	0.860	0.916	0.93
K(t)	-0.425	-0.151	-0.088	-0.073

Table 17.

Observed Bin Proportions, F(t) and K(t) for Moderate Smokers In Smoking Condition

	0-400 ms	400-600 ms	600-800 ms	800-1000 ms
OO				
Observed proportion	0.655	0.217	0.050	0.014
F(t)	0.655	0.872	0.922	0.936
K(t)	-0.423	-0.137	-0.081	-0.066
O _				
Observed proportion	0.619	0.250	0.067	0.021
F(t)	0.619	0.869	0.936	0.957
K(t)	-0.480	-0.140	-0.066	-0.044
_O				
Observed proportion	0.700	0.171	0.053	0.017
F(t)	0.700	0.871	0.924	0.941
K(t)	-0.357	-0.138	-0.079	-0.061

Table 18.

Observed Bin Proportions, F(t) and K(t) for Daily Smokers In Smoking Condition

	0-400 ms	400-600 ms	600-800 ms	800-1000 ms
OO				
Observed proportion	0.735	0.188	0.031	0.017
F(t)	0.735	0.923	0.954	0.971
K(t)	-0.308	-0.080	-0.047	-0.029
O _				
Observed proportion	0.669	0.229	0.052	0.021
F(t)	0.669	0.898	0.950	0.971
K(t)	-0.402	-0.108	-0.051	-0.029
_O				
Observed proportion	0.733	0.187	0.037	0.013
F(t)	0.733	0.920	0.957	0.970
K(t)	-0.311	-0.083	-0.044	-0.031

Table 19.

Observed Bin Proportions, F(t) and K(t) for Non-Smokers In Pseudosmoking Condition

	0-400 ms	400-600 ms	600-800 ms	800-1000 ms
OO				
Observed proportion	0.615	0.205	0.053	0.065
F(t)	0.615	0.820	0.873	0.938
K(t)	-0.486	-0.199	-0.136	-0.064
O _				
Observed proportion	0.565	0.223	0.080	0.040
F(t)	0.565	0.788	0.868	0.908
K(t)	-0.571	-0.238	-0.142	-0.097
_O				
Observed proportion	0.590	0.225	0.063	0.040
F(t)	0.590	0.815	0.878	0.918
K(t)	-0.528	-0.205	-0.130	-0.086

Table 20.

Observed Bin Proportions, F(t) and K(t) for Light Smokers In Pseudosmoking Condition

	0-400 ms	400-600 ms	600-800 ms	800-1000 ms
OO				
Observed proportion	0.668	0.232	0.042	0.022
F(t)	0.668	0.900	0.942	0.964
K(t)	-0.404	-0.105	-0.060	-0.037
O _				
Observed proportion	0.646	0.190	0.074	0.030
F(t)	0.646	0.836	0.910	0.940
K(t)	-0.437	-0.179	-0.094	-0.062
_O				
Observed proportion	0.664	0.184	0.054	0.024
F(t)	0.664	0.848	0.902	0.926
K(t)	-0.410	-0.165	-0.103	-0.077

Table 21.

Observed Bin Proportions, F(t) and K(t) for Moderate Smokers In Pseudosmoking Condition

	0-400 ms	400-600 ms	600-800 ms	800-1000 ms
OO				
Observed proportion	0.624	0.202	0.057	0.034
F(t)	0.624	0.826	0.883	0.917
K(t)	-0.472	-0.191	-0.124	-0.087
O _				
Observed proportion	0.597	0.226	0.067	0.029
F(t)	0.597	0.823	0.89	0.919
K(t)	-0.516	-0.195	-0.117	-0.085
_O				
Observed proportion	0.591	0.212	0.072	0.036
F(t)	0.591	0.803	0.875	0.911
K(t)	-0.526	-0.219	-0.134	-0.093

Table 22.

Observed Bin Proportions, F(t) and K(t) for Daily Smokers In Pseudosmoking Condition

	0-400 ms	400-600 ms	600-800 ms	800-1000 ms
OO				
Observed proportion	0.681	0.212	0.04	0.025
F(t)	0.681	0.893	0.933	0.958
K(t)	-0.384	-0.113	-0.069	-0.043
O _				
Observed proportion	0.621	0.248	0.06	0.023
F(t)	0.621	0.869	0.929	0.952
K(t)	-0.476	-0.140	-0.074	-0.049
_O				
Observed proportion	0.662	0.204	0.048	0.033
F(t)	0.662	0.866	0.914	0.947
K(t)	-0.413	-0.144	-0.090	-0.055

Table 23.

Observed Bin Proportions, Estimated Survivor Functions and Integrated Hazard Functions for Light Smokers/Low Co-Users In No Smoking Condition

	0-400 ms	400-600 ms	600-800 ms	800-1000 ms
XX				
Observed proportion	0.900	0.090	0.010	0.000
Estimated survivor function	0.100	0.010	0.000	0.000
Integrated Hazard Function	2.303	4.605	4.605	4.605
X _				
Observed proportion	0.890	0.080	0.010	0.010
Estimated survivor function	0.110	0.030	0.020	0.010
Integrated Hazard Function	2.207	3.507	3.912	4.605
_ X				
Observed proportion	0.890	0.080	0.010	0.010
Estimated survivor function	0.110	0.030	0.020	0.010
Integrated Hazard Function	2.207	3.507	3.912	4.605
XO				
Observed proportion	0.770	0.180	0.050	0.000
Estimated survivor function	0.230	0.050	0.000	0.000
Integrated Hazard Function	1.470	2.996	2.996	2.996

	0-400 ms	400-600 ms	600-800 ms	800-1000 ms
OX				
Observed proportion	0.630	0.180	0.110	0.050
Estimated survivor function	0.370	0.190	0.080	0.030
Integrated Hazard Function	0.994	1.661	2.526	3.507
OO				
Observed proportion	0.780	0.160	0.030	0.020
Estimated survivor function	0.220	0.060	0.030	0.010
Integrated Hazard Function	1.514	2.813	3.507	4.605
O_				
Observed proportion	0.730	0.190	0.060	0.010
Estimated survivor function	0.270	0.080	0.020	0.010
Integrated Hazard Function	1.309	2.526	3.912	4.605
_O				
Observed proportion	0.780	0.130	0.040	0.030
Estimated survivor function	0.220	0.090	0.050	0.020
Integrated Hazard Function	1.514	2.408	2.996	3.912

Table 24.

Observed Bin Proportions, Estimated Survivor Functions and Integrated Hazard Functions for Light Smokers/Low Co-Users In Smoking Condition

	0-400 ms	400-600 ms	600-800 ms	800-1000 ms
XX				
Observed proportion	0.800	0.120	0.000	0.010
Estimated survivor function	0.200	0.080	0.080	0.070
Integrated Hazard Function	1.609	2.526	2.526	2.659
X _				
Observed proportion	0.750	0.160	0.040	0.000
Estimated survivor function	0.250	0.090	0.050	0.050
Integrated Hazard Function	1.386	2.408	2.996	2.996
_ X				
Observed proportion	0.700	0.200	0.010	0.010
Estimated survivor function	0.300	0.100	0.090	0.080
Integrated Hazard Function	1.204	2.303	2.408	2.526
XO				
Observed proportion	0.700	0.180	0.050	0.020
Estimated survivor function	0.300	0.120	0.070	0.050
Integrated Hazard Function	1.204	2.120	2.659	2.996

	0-400 ms	400-600 ms	600-800 ms	800-1000 ms
OX				
Observed proportion	0.600	0.250	0.050	0.060
Estimated survivor function	0.400	0.150	0.100	0.040
Integrated Hazard Function	0.916	1.897	2.303	3.219
OO				
Observed proportion	0.720	0.160	0.040	0.020
Estimated survivor function	0.280	0.120	0.080	0.060
Integrated Hazard Function	1.273	2.120	2.526	2.813
O_				
Observed proportion	0.640	0.220	0.060	0.030
Estimated survivor function	0.360	0.140	0.080	0.050
Integrated Hazard Function	1.022	1.966	2.526	2.996
_O				
Observed proportion	0.640	0.220	0.060	0.030
Estimated survivor function	0.360	0.140	0.080	0.050
Integrated Hazard Function	1.022	1.966	2.526	2.996

Table 25.

Observed Bin Proportions, Estimated Survivor Functions and Integrated Hazard Functions for Light Smokers/Low Co-Users In Pseudosmoking Condition

	0-400 ms	400-600 ms	600-800 ms	800-1000 ms
XX				
Observed proportion	0.720	0.140	0.040	0.040
Estimated survivor function	0.280	0.140	0.100	0.060
Integrated Hazard Function	1.273	1.966	2.303	2.813
X _				
Observed proportion	0.700	0.110	0.070	0.070
Estimated survivor function	0.300	0.190	0.120	0.050
Integrated Hazard Function	1.204	1.661	2.120	2.996
_ X				
Observed proportion	0.700	0.160	0.060	0.010
Estimated survivor function	0.300	0.140	0.080	0.070
Integrated Hazard Function	1.204	1.966	2.526	2.659
XO				
Observed proportion	0.730	0.190	0.030	0.010
Estimated survivor function	0.270	0.080	0.050	0.040
Integrated Hazard Function	1.309	2.526	2.996	3.219

	0-400 ms	400-600 ms	600-800 ms	800-1000 ms
OX				
Observed proportion	0.510	0.280	0.110	0.050
Estimated survivor function	0.490	0.210	0.100	0.050
Integrated Hazard Function	0.713	1.561	2.303	2.996
OO				
Observed proportion	0.720	0.180	0.040	0.020
Estimated survivor function	0.280	0.100	0.060	0.040
Integrated Hazard Function	1.273	2.303	2.813	3.219
O_				
Observed proportion	0.660	0.170	0.070	0.030
Estimated survivor function	0.340	0.170	0.100	0.070
Integrated Hazard Function	1.079	1.772	2.303	2.659
_O				
Observed proportion	0.660	0.190	0.060	0.030
Estimated survivor function	0.340	0.150	0.090	0.060
Integrated Hazard Function	1.079	1.897	2.408	2.813

Table 26.

Observed Bin Proportions, Estimated Survivor Functions and Integrated Hazard Functions for Light Smokers/High Co-Users In No Smoking Condition

	0-400 ms	400-600 ms	600-800 ms	800-1000 ms
XX				
Observed proportion	0.937	0.050	0.005	0.000
Estimated survivor function	0.063	0.013	0.008	0.008
Integrated Hazard Function	2.765	4.343	4.828	4.828
X _				
Observed proportion	0.929	0.058	0.008	0.003
Estimated survivor function	0.071	0.013	0.005	0.002
Integrated Hazard Function	2.645	4.343	5.298	6.215
_ X				
Observed proportion	0.834	0.142	0.013	0.005
Estimated survivor function	0.166	0.024	0.011	0.006
Integrated Hazard Function	1.796	3.730	4.510	5.116
XO				
Observed proportion	0.868	0.106	0.013	0.003
Estimated survivor function	0.132	0.026	0.013	0.010
Integrated Hazard Function	2.025	3.650	4.343	4.605

	0-400 ms	400-600 ms	600-800 ms	800-1000 ms
OX				
Observed proportion	0.663	0.266	0.053	0.008
Estimated survivor function	0.337	0.071	0.018	0.010
Integrated Hazard Function	1.088	2.645	4.017	4.605
OO				
Observed proportion	0.837	0.134	0.021	0.000
Estimated survivor function	0.163	0.029	0.008	0.008
Integrated Hazard Function	1.814	3.540	4.828	4.828
O_				
Observed proportion	0.750	0.203	0.026	0.005
Estimated survivor function	0.250	0.047	0.021	0.016
Integrated Hazard Function	1.386	3.058	3.863	4.135
_O				
Observed proportion	0.868	0.105	0.013	0.008
Estimated survivor function	0.132	0.027	0.014	0.006
Integrated Hazard Function	2.025	3.612	4.269	5.116

Table 27.

Observed Bin Proportions, Estimated Survivor Functions and Integrated Hazard Functions for Light Smokers/High Co-Users In Smoking Condition

	0-400 ms	400-600 ms	600-800 ms	800-1000 ms
XX				
Observed proportion	0.721	0.147	0.050	0.021
Estimated survivor function	0.279	0.132	0.082	0.061
Integrated Hazard Function	1.277	2.025	2.501	2.797
X _				
Observed proportion	0.739	0.132	0.032	0.016
Estimated survivor function	0.261	0.129	0.097	0.081
Integrated Hazard Function	1.343	2.048	2.333	2.513
_ X				
Observed proportion	0.684	0.171	0.058	0.024
Estimated survivor function	0.316	0.145	0.087	0.063
Integrated Hazard Function	1.152	1.931	2.442	2.765
XO				
Observed proportion	0.742	0.126	0.032	0.042
Estimated survivor function	0.258	0.132	0.100	0.058
Integrated Hazard Function	1.355	2.025	2.303	2.847

	0-400 ms	400-600 ms	600-800 ms	800-1000 ms
OX				
Observed proportion	0.534	0.253	0.095	0.050
Estimated survivor function	0.466	0.213	0.118	0.068
Integrated Hazard Function	0.764	1.546	2.137	2.688
OO				
Observed proportion	0.702	0.203	0.024	0.016
Estimated survivor function	0.298	0.095	0.071	0.055
Integrated Hazard Function	1.211	2.354	2.645	2.900
O_				
Observed proportion	0.639	0.218	0.045	0.029
Estimated survivor function	0.361	0.143	0.098	0.069
Integrated Hazard Function	1.019	1.945	2.323	2.674
_O				
Observed proportion	0.671	0.208	0.050	0.008
Estimated survivor function	0.329	0.121	0.071	0.063
Integrated Hazard Function	1.112	2.112	2.645	2.765

Table 28.

Observed Bin Proportions, Estimated Survivor Functions and Integrated Hazard Functions for Light Smokers/High Co-Users In Pseudosmoking Condition

	0-400 ms	400-600 ms	600-800 ms	800-1000 ms
XX				
Observed proportion	0.776	0.124	0.042	0.021
Estimated survivor function	0.224	0.100	0.058	0.037
Integrated Hazard Function	1.496	2.303	2.847	3.297
X _				
Observed proportion	0.766	0.116	0.039	0.032
Estimated survivor function	0.234	0.118	0.079	0.047
Integrated Hazard Function	1.452	2.137	2.538	3.058
_ X				
Observed proportion	0.732	0.158	0.034	0.026
Estimated survivor function	0.268	0.110	0.076	0.050
Integrated Hazard Function	1.317	2.207	2.577	2.996
XO				
Observed proportion	0.728	0.137	0.071	0.021
Estimated survivor function	0.272	0.135	0.064	0.043
Integrated Hazard Function	1.302	2.002	2.749	3.147

	0-400 ms	400-600 ms	600-800 ms	800-1000 ms
OX				
Observed proportion	0.584	0.234	0.068	0.039
Estimated survivor function	0.416	0.182	0.114	0.075
Integrated Hazard Function	0.877	1.704	2.172	2.590
OO				
Observed proportion	0.703	0.186	0.030	0.014
Estimated survivor function	0.297	0.111	0.081	0.067
Integrated Hazard Function	1.214	2.198	2.513	2.703
O_				
Observed proportion	0.647	0.205	0.068	0.032
Estimated survivor function	0.353	0.148	0.080	0.048
Integrated Hazard Function	1.041	1.911	2.526	3.037
_O				
Observed proportion	0.682	0.176	0.047	0.021
Estimated survivor function	0.318	0.142	0.095	0.074
Integrated Hazard Function	1.146	1.952	2.354	2.604

Table 29.

Observed Bin Proportions, Estimated Survivor Functions and Integrated Hazard Functions for Moderate Smokers/Low Co-Users In No Smoking Condition

	0-400 ms	400-600 ms	600-800 ms	800-1000 ms
XX				
Observed proportion	0.895	0.081	0.010	0.007
Estimated survivor function	0.105	0.024	0.014	0.007
Integrated Hazard Function	2.254	3.730	4.269	4.962
X _				
Observed proportion	0.912	0.067	0.007	0.007
Estimated survivor function	0.088	0.021	0.014	0.007
Integrated Hazard Function	2.430	3.863	4.269	4.962
_ X				
Observed proportion	0.814	0.148	0.024	0.012
Estimated survivor function	0.186	0.038	0.014	0.002
Integrated Hazard Function	1.682	3.270	4.269	6.215
XO				
Observed proportion	0.814	0.143	0.026	0.010
Estimated survivor function	0.186	0.043	0.017	0.007
Integrated Hazard Function	1.682	3.147	4.075	4.962

	0-400 ms	400-600 ms	600-800 ms	800-1000 ms
OX				
Observed proportion	0.757	0.164	0.055	0.012
Estimated survivor function	0.243	0.079	0.024	0.012
Integrated Hazard Function	1.415	2.538	3.730	4.423
OO				
Observed proportion	0.752	0.200	0.021	0.014
Estimated survivor function	0.248	0.048	0.027	0.013
Integrated Hazard Function	1.394	3.037	3.612	4.343
O_				
Observed proportion	0.717	0.214	0.050	0.005
Estimated survivor function	0.283	0.069	0.019	0.014
Integrated Hazard Function	1.262	2.674	3.963	4.269
_O				
Observed proportion	0.774	0.195	0.019	0.007
Estimated survivor function	0.226	0.031	0.012	0.005
Integrated Hazard Function	1.487	3.474	4.423	5.298

Table 30.

Observed Bin Proportions, Estimated Survivor Functions and Integrated Hazard Functions for Moderate Smokers/Low Co-Users In Smoking Condition

	0-400 ms	400-600 ms	600-800 ms	800-1000 ms
XX				
Observed proportion	0.798	0.129	0.021	0.017
Estimated survivor function	0.202	0.073	0.052	0.035
Integrated Hazard Function	1.599	2.617	2.957	3.352
X _				
Observed proportion	0.757	0.140	0.040	0.014
Estimated survivor function	0.243	0.103	0.063	0.049
Integrated Hazard Function	1.415	2.273	2.765	3.016
_ X				
Observed proportion	0.698	0.198	0.036	0.012
Estimated survivor function	0.302	0.104	0.068	0.056
Integrated Hazard Function	1.197	2.263	2.688	2.882
XO				
Observed proportion	0.719	0.155	0.071	0.031
Estimated survivor function	0.281	0.126	0.055	0.024
Integrated Hazard Function	1.269	2.071	2.900	3.730

	0-400 ms	400-600 ms	600-800 ms	800-1000 ms
OX				
Observed proportion	0.598	0.243	0.067	0.048
Estimated survivor function	0.402	0.159	0.092	0.044
Integrated Hazard Function	0.911	1.839	2.386	3.124
OO				
Observed proportion	0.660	0.231	0.050	0.012
Estimated survivor function	0.340	0.109	0.059	0.047
Integrated Hazard Function	1.079	2.216	2.830	3.058
O_				
Observed proportion	0.595	0.264	0.062	0.029
Estimated survivor function	0.405	0.141	0.079	0.050
Integrated Hazard Function	0.904	1.959	2.538	2.996
_O				
Observed proportion	0.717	0.183	0.050	0.010
Estimated survivor function	0.283	0.100	0.050	0.040
Integrated Hazard Function	1.262	2.303	2.996	3.219

Table 31.

Observed Bin Proportions, Estimated Survivor Functions and Integrated Hazard Functions for Moderate/Low Co-Users In Pseudosmoking Condition

	0-400 ms	400-600 ms	600-800 ms	800-1000 ms
XX				
Observed proportion	0.764	0.145	0.026	0.019
Estimated survivor function	0.236	0.091	0.065	0.046
Integrated Hazard Function	1.444	2.397	2.733	3.079
X _				
Observed proportion	0.738	0.117	0.045	0.029
Estimated survivor function	0.262	0.145	0.100	0.071
Integrated Hazard Function	1.339	1.931	2.303	2.645
_ X				
Observed proportion	0.664	0.202	0.050	0.033
Estimated survivor function	0.336	0.134	0.084	0.051
Integrated Hazard Function	1.091	2.010	2.477	2.976
XO				
Observed proportion	0.640	0.198	0.064	0.036
Estimated survivor function	0.360	0.162	0.098	0.062
Integrated Hazard Function	1.022	1.820	2.323	2.781

	0-400 ms	400-600 ms	600-800 ms	800-1000 ms
OX				
Observed proportion	0.550	0.245	0.071	0.048
Estimated survivor function	0.450	0.205	0.134	0.086
Integrated Hazard Function	0.799	1.585	2.010	2.453
OO				
Observed proportion	0.581	0.250	0.067	0.024
Estimated survivor function	0.419	0.169	0.102	0.078
Integrated Hazard Function	0.870	1.778	2.283	2.551
O_				
Observed proportion	0.605	0.238	0.067	0.026
Estimated survivor function	0.395	0.157	0.090	0.064
Integrated Hazard Function	0.929	1.852	2.408	2.749
_O				
Observed proportion	0.593	0.214	0.081	0.036
Estimated survivor function	0.407	0.193	0.112	0.076
Integrated Hazard Function	0.899	1.645	2.189	2.577

Table 32.

Observed Bin Proportions, Estimated Survivor Functions and Integrated Hazard Functions for Daily Smokers/Low Co-Users In No Smoking Condition

	0-400 ms	400-600 ms	600-800 ms	800-1000 ms
XX				
Observed proportion	0.898	0.090	0.005	0.000
Estimated survivor function	0.102	0.012	0.007	0.007
Integrated Hazard Function	2.283	4.423	4.962	4.962
X _				
Observed proportion	0.912	0.062	0.012	0.007
Estimated survivor function	0.088	0.026	0.014	0.007
Integrated Hazard Function	2.430	3.650	4.269	4.962
_ X				
Observed proportion	0.833	0.143	0.012	0.007
Estimated survivor function	0.167	0.024	0.012	0.005
Integrated Hazard Function	1.790	3.730	4.423	5.298
XO				
Observed proportion	0.800	0.169	0.017	0.007
Estimated survivor function	0.200	0.031	0.014	0.007
Integrated Hazard Function	1.609	3.474	4.269	4.962

	0-400 ms	400-600 ms	600-800 ms	800-1000 ms
OX				
Observed proportion	0.660	0.250	0.060	0.017
Estimated survivor function	0.340	0.090	0.030	0.013
Integrated Hazard Function	1.079	2.408	3.507	4.343
OO				
Observed proportion	0.807	0.160	0.024	0.000
Estimated survivor function	0.193	0.033	0.009	0.009
Integrated Hazard Function	1.645	3.411	4.711	4.711
O_				
Observed proportion	0.721	0.217	0.043	0.005
Estimated survivor function	0.279	0.062	0.019	0.014
Integrated Hazard Function	1.277	2.781	3.963	4.269
_O				
Observed proportion	0.829	0.119	0.029	0.014
Estimated survivor function	0.171	0.052	0.023	0.009
Integrated Hazard Function	1.766	2.957	3.772	4.711

Table 33.

Observed Bin Proportions, Estimated Survivor Functions and Integrated Hazard Functions for Daily Smokers/Low Co-Users In Smoking Condition

	0-400 ms	400-600 ms	600-800 ms	800-1000 ms
XX				
Observed proportion	0.831	0.107	0.033	0.010
Estimated survivor function	0.169	0.062	0.029	0.019
Integrated Hazard Function	1.778	2.781	3.540	3.963
X _				
Observed proportion	0.826	0.129	0.021	0.005
Estimated survivor function	0.174	0.045	0.024	0.019
Integrated Hazard Function	1.749	3.101	3.730	3.963
_ X				
Observed proportion	0.724	0.171	0.045	0.036
Estimated survivor function	0.276	0.105	0.060	0.024
Integrated Hazard Function	1.287	2.254	2.813	3.730
XO				
Observed proportion	0.752	0.160	0.040	0.017
Estimated survivor function	0.248	0.088	0.048	0.031
Integrated Hazard Function	1.394	2.430	3.037	3.474

	0-400 ms	400-600 ms	600-800 ms	800-1000 ms
OX				
Observed proportion	0.562	0.290	0.071	0.031
Estimated survivor function	0.438	0.148	0.077	0.046
Integrated Hazard Function	0.826	1.911	2.564	3.079
OO				
Observed proportion	0.710	0.207	0.029	0.021
Estimated survivor function	0.290	0.083	0.054	0.033
Integrated Hazard Function	1.238	2.489	2.919	3.411
O_				
Observed proportion	0.631	0.255	0.060	0.024
Estimated survivor function	0.369	0.114	0.054	0.030
Integrated Hazard Function	0.997	2.172	2.919	3.507
_O				
Observed proportion	0.724	0.205	0.026	0.014
Estimated survivor function	0.276	0.071	0.045	0.031
Integrated Hazard Function	1.287	2.645	3.101	3.474

Table 34.

Observed Bin Proportions, Estimated Survivor Functions and Integrated Hazard Functions for Daily Smokers/Low Co-Users In Pseudosmoking Condition

	0-400 ms	400-600 ms	600-800 ms	800-1000 ms
XX				
Observed proportion	0.738	0.167	0.031	0.010
Estimated survivor function	0.262	0.095	0.064	0.054
Integrated Hazard Function	1.339	2.354	2.749	2.919
X _				
Observed proportion	0.719	0.143	0.052	0.024
Estimated survivor function	0.281	0.138	0.086	0.062
Integrated Hazard Function	1.269	1.981	2.453	2.781
_ X				
Observed proportion	0.662	0.236	0.050	0.029
Estimated survivor function	0.338	0.102	0.052	0.023
Integrated Hazard Function	1.085	2.283	2.957	3.772
XO				
Observed proportion	0.705	0.183	0.043	0.014
Estimated survivor function	0.295	0.112	0.069	0.055
Integrated Hazard Function	1.221	2.189	2.674	2.900

	0-400 ms	400-600 ms	600-800 ms	800-1000 ms
OX				
Observed proportion	0.564	0.238	0.102	0.043
Estimated survivor function	0.436	0.198	0.096	0.053
Integrated Hazard Function	0.830	1.619	2.343	2.937
OO				
Observed proportion	0.657	0.231	0.045	0.026
Estimated survivor function	0.343	0.112	0.067	0.041
Integrated Hazard Function	1.070	2.189	2.703	3.194
O_				
Observed proportion	0.602	0.262	0.069	0.017
Estimated survivor function	0.398	0.136	0.067	0.050
Integrated Hazard Function	0.921	1.995	2.703	2.996
_O				
Observed proportion	0.655	0.205	0.060	0.019
Estimated survivor function	0.345	0.140	0.080	0.061
Integrated Hazard Function	1.064	1.966	2.526	2.797

Table 35.

Observed Bin Proportions, Estimated Survivor Functions and Integrated Hazard Functions for Moderate and Daily Smokers/High Co-Users In No Smoking Condition

	0-400 ms	400-600 ms	600-800 ms	800-1000 ms
XX				
Observed proportion	0.917	0.083	0.000	0.000
Estimated survivor function	0.083	0.000	0.000	0.000
Integrated Hazard Function	2.489	2.489	2.489	2.489
X _				
Observed proportion	0.925	0.050	0.025	0.000
Estimated survivor function	0.075	0.025	0.000	0.000
Integrated Hazard Function	2.590	3.689	3.689	3.689
_ X				
Observed proportion	0.867	0.108	0.017	0.008
Estimated survivor function	0.133	0.025	0.008	0.000
Integrated Hazard Function	2.017	3.689	4.828	4.828
XO				
Observed proportion	0.817	0.158	0.025	0.000
Estimated survivor function	0.183	0.025	0.000	0.000
Integrated Hazard Function	1.698	3.689	3.689	3.689

	0-400 ms	400-600 ms	600-800 ms	800-1000 ms
OX				
Observed proportion	0.658	0.250	0.050	0.025
Estimated survivor function	0.342	0.092	0.042	0.017
Integrated Hazard Function	1.073	2.386	3.170	4.075
OO				
Observed proportion	0.750	0.158	0.067	0.008
Estimated survivor function	0.250	0.092	0.025	0.017
Integrated Hazard Function	1.386	2.386	3.689	4.075
O_				
Observed proportion	0.808	0.133	0.042	0.008
Estimated survivor function	0.192	0.059	0.017	0.009
Integrated Hazard Function	1.650	2.830	4.075	4.711
_O				
Observed proportion	0.808	0.125	0.058	0.008
Estimated survivor function	0.192	0.067	0.009	0.001
Integrated Hazard Function	1.650	2.703	4.711	6.908

Table 36.

Observed Bin Proportions, Estimated Survivor Functions and Integrated Hazard Functions for Moderate and Daily Smokers/High Co-Users In Smoking Condition

	0-400 ms	400-600 ms	600-800 ms	800-1000 ms
XX				
Observed proportion	0.833	0.075	0.017	0.042
Estimated survivor function	0.167	0.092	0.075	0.033
Integrated Hazard Function	1.790	2.386	2.590	3.411
X _				
Observed proportion	0.750	0.108	0.058	0.080
Estimated survivor function	0.250	0.142	0.084	0.004
Integrated Hazard Function	1.386	1.952	2.477	5.521
_ X				
Observed proportion	0.750	0.167	0.050	0.000
Estimated survivor function	0.250	0.083	0.033	0.033
Integrated Hazard Function	1.386	2.489	3.411	3.411
XO				
Observed proportion	0.767	0.117	0.025	0.017
Estimated survivor function	0.233	0.116	0.091	0.074
Integrated Hazard Function	1.457	2.154	2.397	2.604

	0-400 ms	400-600 ms	600-800 ms	800-1000 ms
OX				
Observed proportion	0.625	0.192	0.067	0.042
Estimated survivor function	0.375	0.183	0.116	0.074
Integrated Hazard Function	0.981	1.698	2.154	2.604
OO				
Observed proportion	0.717	0.133	0.050	0.008
Estimated survivor function	0.283	0.150	0.100	0.092
Integrated Hazard Function	1.262	1.897	2.303	2.386
O_				
Observed proportion	0.733	0.142	0.067	0.033
Estimated survivor function	0.267	0.125	0.058	0.025
Integrated Hazard Function	1.321	2.079	2.847	3.689
_O				
Observed proportion	0.717	0.142	0.033	0.017
Estimated survivor function	0.283	0.141	0.108	0.091
Integrated Hazard Function	1.262	1.959	2.226	2.397

Table 37.

Observed Bin Proportions, Estimated Survivor Functions and Integrated Hazard Functions for Moderate and Daily Smokers/High Co-Users In Pseudosmoking Condition

	0-400 ms	400-600 ms	600-800 ms	800-1000 ms
XX				
Observed proportion	0.750	0.167	0.008	0.008
Estimated survivor function	0.250	0.083	0.075	0.067
Integrated Hazard Function	1.386	2.489	2.590	2.703
X _				
Observed proportion	0.733	0.092	0.050	0.017
Estimated survivor function	0.267	0.175	0.125	0.108
Integrated Hazard Function	1.321	1.743	2.079	2.226
_ X				
Observed proportion	0.700	0.150	0.033	0.017
Estimated survivor function	0.300	0.150	0.117	0.100
Integrated Hazard Function	1.204	1.897	2.146	2.303
XO				
Observed proportion	0.683	0.142	0.058	0.033
Estimated survivor function	0.317	0.175	0.117	0.084
Integrated Hazard Function	1.149	1.743	2.146	2.477

	0-400 ms	400-600 ms	600-800 ms	800-1000 ms
OX				
Observed proportion	0.658	0.217	0.050	0.025
Estimated survivor function	0.342	0.125	0.075	0.050
Integrated Hazard Function	1.073	2.079	2.590	2.996
OO				
Observed proportion	0.750	0.100	0.033	0.058
Estimated survivor function	0.250	0.150	0.117	0.059
Integrated Hazard Function	1.386	1.897	2.146	2.830
O_				
Observed proportion	0.683	0.167	0.033	0.025
Estimated survivor function	0.317	0.150	0.117	0.092
Integrated Hazard Function	1.149	1.897	2.146	2.386
_O				
Observed proportion	0.617	0.225	0.058	0.025
Estimated survivor function	0.383	0.158	0.100	0.075
Integrated Hazard Function	0.960	1.845	2.303	2.590

AGNES MASSAK-WAINMAN

Curriculum Vitae

EDUCATION

2004-2011 **Doctor of Philosophy, Clinical Psychology**
University of Western Ontario, London, Ontario
Advisor: Dr. Riley Hinson

Dissertation: Smoking as an Automatic Process: Using Capacity Coefficients Among Distinct Typologies of Smokers

2002 – 2004 **Master of Arts, Clinical Psychology**
University of Western Ontario, London, Ontario
Advisor: Dr. Riley Hinson

Thesis: Encoding and Retrieval of Novel Information Using Smoking as a Secondary Task to Investigate the Automaticity of Smoking

1997-2001 **Honour Bachelor of Science, With High Distinction**
University of Toronto
Advisor: Dr. Janet Polivy

Thesis: Food Availability and Variety: The Effects on Intake in Restrained and Unrestrained Eaters.

SUPERVISED CLINICAL EXPERIENCE

Sept. 2009-Aug. 2010 **London Clinical Psychology Internship Consortium**

March 2010-August 2010
Service: Consultation-Liaison Psychiatry Service
Supervisor: Dr. Naomi Wiesenthal
Clinical Duties: Assessment and follow-up of medically ill inpatients referred for mental health concerns. Involves working on a multidisciplinary team including psychology, psychiatry and nursing, and consulting with physicians and allied health professionals.

Service: Cardiac Rehabilitation and Secondary Prevention Program
Supervisor: Drs. Peter Prior and Judith Francis
Clinical Duties: Assessment and psychological treatment for patients with a history of cardiovascular disease. Also involves co-leading weekly smoking cessation group for patients and their family members.

Service: Operational Stress Injury Clinic, Parkwood Hospital
Supervisor: Dr. Maya Roth
Clinical Duties: Psychological treatment of post-traumatic stress disorder in military service people (currently serving and veterans).

Service: Epilepsy Unit and Neurology Outpatient Clinic, University Hospital
Supervisor: Dr. Paul Derry
Clinical Duties: Inpatient assessments of individuals within the Epilepsy Unit. Additional duties included assessment and psychological treatment of outpatients referred by neurologists for mental health concerns.

Service: Reproductive Endocrinology and Infertility Service
Supervisor: Dr. Christopher Newton
Clinical Duties: Assessment of couples entering fertility treatment program. Focus of assessment included coping with the stressors of infertility and treatment protocol. Also provided brief crisis intervention for individual within the program who presented with mental health concerns.

May 2007- **Spinal Cord Injury Rehabilitation Program**
Dec. 2007 Parkwood Hospital, St. Joseph's Hospital
Supervisor: Steve Orenczuk, Psy. D., C. Psych
Clinical Duties: Planned and conducted inpatient psychological assessments and outpatient assessments with clients with spinal cord injuries using clinical interviews and standardized psychological tests. Provided weekly individual psychotherapy to outpatients and inpatients.

August 2006- **Behavioral Medicine Service**
April 2007 London Health Science Centre, London, Ontario
Supervisor: Felicia Otchet, Ph.D., C. Psych
Clinical Duties: Planned and conducted outpatient psychological assessments using clinical interviews, personality tests, and specific standardized psychological tests. Provided weekly individual and group psychotherapy to individuals with chronic pain.

May. 2006- **Adult Ambulatory Outpatient Services**
Feb. 2007 London Health Science Centre, London, Ontario
Supervisor: Elizabeth Werth, Ph.D., C. Psych
Clinical Duties: Provided individual long-term psychotherapy to individuals from a psychodynamic orientation.

Feb. 2006- **Private Practice**
Feb. 2007 London, Ontario
Supervisor: Kate Partridge, Ph.D., C. Psych
Clinical Duties: Received training in leading mindfulness meditation groups for individuals seeking stress reduction management skills. Co-led two groups in the practice of mindfulness meditation in a university population setting.

Sept. 2005- **Acquired Brain Injury Program**
May 2006 Parkwood Hospital, London, Ontario
Supervisor: Margaret Weiser, Ph.D., C. Psych
Clinical Duties: Provided individual psychotherapy to clients with sustained brain injuries. Conducted clinical interviews with clients with brain injuries, assessing current functioning. Consulted with other health care professionals regarding treatment planning.

May 2005- **Regional Mental Health Care**
January 2006 St. Thomas, Ontario
Supervisor: Fred Meek, Ph.D., C. Psych
Clinical Duties: Provided individual psychotherapy to clients with varied mental health concerns, and conducted comprehensive assessments.

Sept. 2004- **Student Development Centre Counselling Services**
Aug. 2005 University of Western Ontario
London, Ontario
Supervisor: Kathy Dance, Ph.D., C. Psych
Clinical Duties: Conducted intake assessments, administered standardized personality and symptom-focused tests, provided brief intervention for individuals in crisis, and provided weekly psychotherapy for young adults with a broad range of difficulties.

May 2004- **Regional Mental Health Care**
Nov. 2004 St. Thomas, Ontario
Supervisor: Charles Nelson, Ph.D., C. Psych
Clinical Duties: Conducted comprehensive assessments and provided feedback to clients who were referred for a range of mental health concerns.

RESEARCH EMPLOYMENT POSITIONS

2004-2008 **Research Assistant**
Centre for Addiction and Mental Health
London, Ontario
Supervisor: Kathryn Graham, Ph.D.
Research Project: Gender, Alcohol and Culture: An International Study

Responsibilities: Study of 14,000 Canadians and their alcohol and drug use, and other variables, such as depression, partner violence, and motives for drinking. Responsibilities included literature reviews, data analysis, and manuscript preparation.

2003 – 2004 **Research Assistant**
Brain and Behaviour Program, The Hospital for Sick Children
Toronto, Ontario
Supervisor: Dr. Joseph Beitchman
Research Project: Ottawa Language Study

Responsibilities: A 20-year longitudinal study examining speech and language development and its relationship to academic, cognitive and behavioural development. Responsibilities included data entry and data base management.

2001-2002 **Concurrent Disorders Program, Eating Disorders and Substance Use Unit, Centre for Addiction and Mental Health**
Toronto, Ontario
Supervisor: Dr. Christine Courbasson
Research Project: Dialectical Behaviour Therapy as a Treatment for clients with a substance use problem and an eating disorder
Responsibilities: Assessed clients on a monthly basis, data entry, database management.

2000-2001 **Research Assistant**
Child and Family Studies Unit, Centre for Addiction and Mental Health
Toronto, Ontario
Supervisor: Dr. Joseph Beitchman

Research Project: Ottawa Language Study
Responsibilities: A 20-year longitudinal study examining speech and language development and its relationship to academic, cognitive and behavioural development. Responsibilities included maintaining contact with participants, data analysis, and manuscript preparation.

PUBLICATIONS

Massak, A., Graham, K. (2009). The relationship between depression, alcohol use disorder and coping motives for drinking among a national sample of Canadian men and women. In Leo Sher (Ed.) *Comorbidity of Depression and Alcohol Use Disorders.* Nova Science Publishers: New York.

Massak, A., & Graham, K. (2008). Is the smoking-depression relationship confounded by alcohol consumption? An analysis by gender. *Nicotine and Tobacco Research, 10*, 1231-1243.

Graham, K., & **Massak, A.** (2007). Gender Differences in the Moderating Effect of Use of Antidepressants Medications on the Relationship between Alcohol Use and Depression in a General Population Sample. *Canadian Medical Association Journal.*

Graham, K., **Massak, A**., Demers, A., & Rehm, J. (2007). The Effect of Measurement, Timeframe and Gender on the Association between Depression and Alcohol Consumption. Analysis of the GENACIS Canada Survey. *Alcoholism: Clinical and Experimental Research.*

Beitchman, J. H., Adlaf, E., Atkinson, L., Douglas, L., **Massak, A**., & Kenaszchuk, C. (2005). Psychiatric and Substance Use Disorders in Late Adolescence: The Role of Risk and Perceived Social Support. *The American Journal on Addiction, 14*, 124-138.

CONFERENCE PRESENTATIONS

Graham, K., Bernards, S., & **Massak, A**. *GENACIS: Drinking Across Cultures and its Relation to Depression.* Presentation at The Institute of Survey Research, Toronto, Ontario. November, 2004.

Beitchman, J. H., Kennedy, J., Atkinson, L., Quist, J., Pozzulo, J., Seto, M., Espinet, S., Douglas, L., **Massak, A**. *A Pilot Study of Serotonin System Genes and Aggressive Behavior in Children: Preliminary Findings*. Poster presentation: Society for the Study of Behavioral Phenotypes, 6th International Meeting, San Servolo, Venice. October 12-14, 2000.