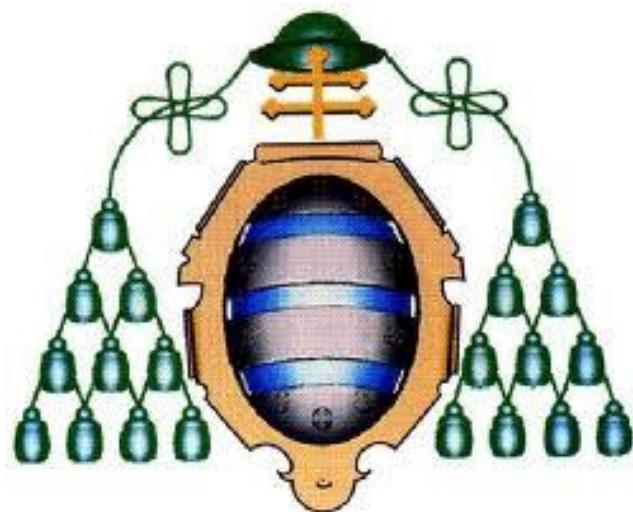


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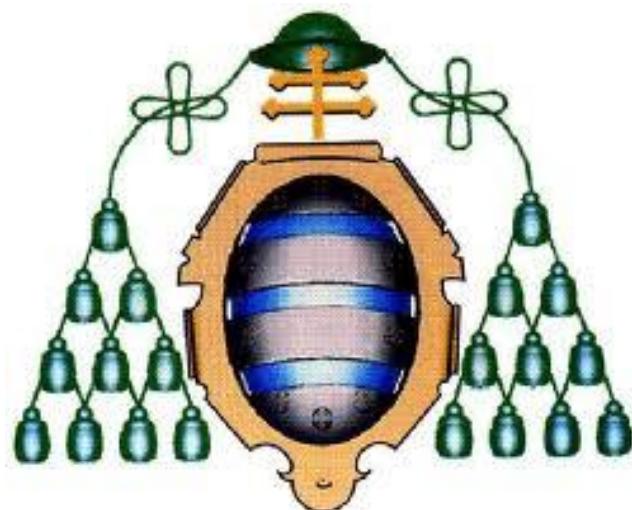
DEPARTAMENTO DE PSICOLOGÍA
PROGRAMA DE DOCTORADO DE PSICOLOGÍA

EVALUACIÓN DE LA IMPULSIVIDAD
MEDIANTE LA APLICACIÓN DE LA TAREA
DE DESCUENTO POR DEMORA EN SUJETOS
DEPENDIENTES DE SUSTANCIAS

Sara Weidberg López

Oviedo, 2015

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RESUMEN DEL CONTENIDO DE TESIS DOCTORAL

1.- Título de la Tesis	
Español/Otro Idioma: Evaluación de la impulsividad mediante la aplicación de la tarea de descuento por demora en sujetos dependientes de sustancias	Inglés: Assessment of impulsivity through the application of the delay discounting task among substance dependent individuals
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Programa de Doctorado: Psicología	
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RESUMEN (en español)

El término impulsividad se usa de forma genérica en el campo de la Psicología para referirse a conductas que se llevan a cabo con poca o inadecuada reflexión. La impulsividad ha demostrado ser un factor común a muchos trastornos psicopatológicos, como el trastorno bipolar, el trastorno de conducta alimentaria, la conducta suicida, y la dependencia de diferentes sustancias. El descuento por demora constituye un índice conductual de impulsividad, en particular, de toma de decisiones impulsiva, que se define como la velocidad a la que un reforzador pierde su valor a medida que se incrementa el tiempo para recibirla. Un gran número de estudios ha demostrado que el descuento por demora es un fenómeno fundamental a la hora de explicar las conductas de uso de drogas. Sin embargo, existen varios interrogantes todavía no resueltos por parte de la investigación previa. Esta Tesis Doctoral pretende esclarecer algunas de estas cuestiones aún pendientes. Los objetivos de la presente Tesis Doctoral son: (1) comparar las tasas de descuento por demora de sujetos dependientes de diversas sustancias y sujetos controles no dependientes, (2) analizar si el descuento por demora se modifica en función del estatus de consumo en fumadores que reciben un tratamiento para dejar de fumar, (3) evaluar los efectos principales e interactivos de la sintomatología depresiva y los cambios en el estatus de consumo de tabaco sobre las tasas de descuento por demora y (4) evaluar el efecto diferencial de un componente de Manejo de Contingencias (MC) añadido a un tratamiento para dejar de fumar sobre las tasas de descuento por demora. Los resultados mostraron que el tipo de sustancia de la que los individuos dependen tiene una influencia determinante sobre las tasas de



descuento por demora. Por otro lado, las altas tasas de descuento por demora observadas entre la población fumadora pueden reducirse con la abstinencia a largo plazo. Además, las reducciones en el descuento por demora producidas por la abstinencia del tabaco se encuentran acentuadas en los individuos con sintomatología depresiva. Por último, el MC no está asociado de forma robusta con cambios en el descuento por demora. Estos resultados han de ser tomados con cautela, ya que los estudios realizados cuentan con ciertas limitaciones, como el empleo de reforzadores hipotéticos en el procedimiento de descuento por demora, el uso de la medida punto de prevalencia para definir abstinencia en contraposición a la medida de abstinencia continuada, o los tamaños muestrales relativamente pequeños de algunos de los estudios. Más allá de estas limitaciones, los resultados de esta Tesis Doctoral aportan evidencia de que el descuento por demora es una variable estado que puede modificarse y que debería ser considerada en los protocolos de evaluación de la drogodependencia.

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The term impulsivity is used in a generic way within the psychology field to allude to behaviors that are performed with little or inadequate forethought. Impulsivity has proved to be a common factor presented in a wide range of psychopathological disorders, such as bipolar disorder, eating disorder, suicidal behavior, and drug dependence. Delay discounting is a behavioral index of impulsivity, particularly of impulsive decision making, which describes the rate at which a reinforcer loses value as the delay to its receipt increases. Numerous studies have shown that delay discounting is a key phenomenon in order to explain drug use. However, there are still several questions that previous research has not solved yet. This Doctoral Thesis tries to clarify some of these pending issues. The aims of the present Doctoral Thesis are: (1) to compare delay discounting rates among individuals dependent on different substances and non dependent controls, (2) to assess whether delay discounting change as a function of smoking status among smokers who receive a treatment to quit smoking, (3) to assess the main and interactive effects of depressive symptomatology and changes in smoking status on delay discounting, and (4) to analyze the differential effect of a



Contingency Management (CM) component added to a treatment to quit smoking on delay discounting rates. Results showed that the type of substance which the individuals depend on has a fundamental influence on delay discounting. On the other hand, high delay discounting rates observed among smoking population can be reduced by the achievement of long term abstinence. Moreover, delay discounting reductions associated with smoking abstinence are enhanced among individuals with depressive symptoms. Lastly, CM is not robustly associated with delay discounting changes. These results should be taken into account with caution because the studies have certain limitations such as the application of hypothetical rewards in the delay discounting procedure, the use of a point prevalence measure to define abstinence as opposed to a continuous abstinence measure, or the small sample sizes of some of the studies. Beyond these limitations, the results of this Doctoral Thesis provide evidence that delay discounting is a state variable that can be modified and that should be considered in the assessment protocols of drug dependence.

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A mis padres, a mi hermano y a Álex

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RESUMEN

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The term impulsivity is used in a generic way within the psychology field to allude to behaviors that are performed with little or inadequate forethought. Impulsivity has proved to be a common factor presented in a wide range of psychopathological disorders, such as bipolar disorder, eating disorder, suicidal behavior, and drug dependence. Delay discounting is a behavioral index of impulsivity, particularly of impulsive decision making, which describes the rate at which a reinforcer loses value as the delay to its receipt increases. Numerous studies have shown that delay discounting is a key phenomenon in order to explain drug use. However, there are still several questions that previous research has not solved yet. This Doctoral Thesis tries to clarify some of these pending issues. The aims of the present Doctoral Thesis are: (1) to compare delay discounting rates among individuals dependent on different substances and non dependent controls, (2) to assess whether delay discounting change as a function of smoking status among smokers who receive a treatment to quit smoking, (3) to assess the main and interactive effects of depressive symptomatology and changes in smoking status on delay discounting, and (4) to analyze the differential effect of a Contingency Management (CM) component added to a treatment to quit smoking on delay discounting rates. Results showed that the type of substance which the individuals depend on has a fundamental influence on delay discounting. On the other hand, high delay discounting rates observed among smoking population can be reduced by the achievement of long term abstinence. Moreover, delay discounting reductions associated with smoking abstinence are enhanced among individuals with depressive symptoms. Lastly, CM is not robustly associated with delay discounting changes. These results should be taken into account with caution because the studies have certain limitations such as the application of hypothetical rewards in the delay discounting procedure, the use of a point prevalence measure to define abstinence as opposed to a continuous abstinence measure, or the small sample sizes of some of the studies. Beyond these limitations, the results of this Doctoral Thesis provide evidence that delay discounting is a state variable that can be modified and that should be considered in the assessment protocols of drug dependence.

1. INTRODUCCIÓN

1.1. Definición y componentes del constructo de impulsividad

La impulsividad es un aspecto de la conducta que influye en la vida diaria de las personas. El hecho de que la mayor parte de las personas hayan actuado de manera impulsiva en el algún momento de sus vidas (por ejemplo, comprando algo que no necesitan o comiendo más de lo quisieran) hace que este término sea ampliamente usado en el ámbito de lo coloquial (Madden y Johnson, 2010). Sin embargo, aunque parezca fácil identificar ejemplos de conductas impulsivas, es considerablemente más difícil definir este término de forma precisa. En la revisión clásica de Evenden (1999), se recogían hasta 28 términos diferentes que los investigadores habían usado para identificar diversos aspectos del concepto de impulsividad. El mismo Evenden (1999) define el concepto de impulsividad como una acción pobremente concebida, expresada de forma prematura y excesivamente arriesgada o inapropiada para una situación y que suele conllevar resultados no deseados. Otras definiciones de impulsividad incluyen una acción rápida sin reflexión o juicio consciente (Hinslie y Shatzky, 1940), una conducta sin pensamiento adecuado (Smith, 1952), la tendencia de una persona a actuar con menor reflexión en comparación con la mayoría de las personas de igual capacidad y conocimiento (Dickman, 1993), y la predisposición a tener reacciones rápidas y no planificadas a estímulos internos o externos sin tener en cuenta las consecuencias negativas para uno mismo ni para otros (Moeller, Barratt, Dougherty, Schmitz, y Swann, 2001). A pesar de que la literatura previa señala algunos componentes adaptativos y funcionales de la impulsividad (Dickman, 1990), lo cierto es que la mayoría de las definiciones sobre este constructo incluyen algún aspecto disfuncional o desadaptativo asociado con acciones criminales y/o violentas, dañino para uno mismo o inapropiado dentro de los estándares sociales (de Wit, 2009; Verdejo-García, Lawrence, y Clark, 2008).

La investigación en impulsividad ha estado históricamente limitada por una falta de consistencia tanto a la hora de definir el constructo como a la hora de evaluarlo. Esto ha provocado que exista un acuerdo creciente sobre la naturaleza multidimensional de este constructo (de Wit, 2009; Evenden, 1999), así como el hecho de que sea posible que diferentes medidas de impulsividad estén evaluando diferentes componentes subyacentes (Swann, Bjork, Moeller, y Dougherty, 2002). Los tres componentes

subyacentes más comúnmente identificados son los fallos atencionales (impulsividad atencional), la falta de inhibición (impulsividad motora) y la insensibilidad a las consecuencias (impulsividad cognitiva) (Crews y Boettiger, 2009; de Wit, 2009; Patton, Stanford, y Barratt, 1995). No obstante, tampoco existe un consenso acerca del número y del tipo de componentes que subyacen al constructo de impulsividad, que varían en función del análisis realizado a la hora de identificarlos y de los cuestionarios de evaluación empleados. Whiteside y Lyman (2001) identifican cuatro factores de personalidad mediante análisis factorial en el que se emplearon cuestionarios de autoinforme: urgencia, falta de premeditación, falta de perseverancia y búsqueda de sensaciones. Sin embargo, E. Miller, Joseph, y Tudway (2004) describen tres elementos a través de un análisis de componentes principales en el que también se emplearon cuestionarios de autoinforme: impulsividad no planeada y disfuncional, atrevimiento y respuesta al refuerzo. Es importante mencionar que estos dos últimos estudios encontraron diferentes componentes del constructo de impulsividad incluso cuando algunos de los cuestionarios empleados en ambos estudios eran los mismos. Esta falta de consenso también se observa en los estudios que emplean tanto pruebas de autoinforme como pruebas conductuales o de laboratorio. Mientras que Reynolds, Ortengren, Richards, y de Wit (2006) aislaron 2 componentes mediante un análisis de componentes principales (desinhibición impulsiva y toma de decisiones impulsiva), un estudio posterior en el que también se realizó un análisis de componentes principales aisló cinco: activación conductual autoinformada, compulsividad autoinformada y sensibilidad al castigo/refuerzo, impulsividad autoinformada, descuento temporal, y asunción de riesgo (Meda et al., 2009). En resumen, a pesar de que existe un consenso sobre el carácter multifactorial de la impulsividad, la investigación previa sobre los diferentes componentes o factores que la conforman no es concluyente.

1.2. Evaluación del constructo de impulsividad

Los instrumentos de evaluación del constructo de impulsividad se pueden clasificar en dos tipos: cuestionarios de autoinforme y pruebas conductuales o de laboratorio. A continuación se describirán las pruebas de evaluación más comúnmente usadas de cada tipo.

1.2.1. Cuestionarios de autoinforme

A lo largo del tiempo se han desarrollado diferentes cuestionarios de autoinforme diseñados para evaluar distintas características del constructo de impulsividad. Uno de los cuestionarios más antiguos y empleados es la Escala de Impulsividad de Barratt (BIS; Barratt, 1959), que originalmente constaba de 44 ítems en formato verdadero-falso. Tras varias modificaciones de la escala, la Escala de Impulsividad de Barratt -11 (BIS-11; Patton, et al., 1995) representa el último de esfuerzo de Barrat y colaboradores de medir el constructo de impulsividad de forma ortogonal al de ansiedad. La escala BIS-11 consta de 30 ítems medidos en una escala Likert de 4 puntos que oscilan desde 1 (*raramente/nunca*) hasta 4 (*casi siempre*), sin que exista un ítem neutral disponible. Está formada por tres subescalas denominadas impulsividad motora, impulsividad cognitiva e impulsividad no planificada. Los coeficientes de consistencia interna del BIS-11 oscilan entre 0,79 y 0,83 hallados entre grupos de estudiantes universitarios, pacientes psiquiátricos hospitalizados y reclusos (Patton, et al., 1995).

Otro de los cuestionarios comúnmente empleados es el Cuestionario de Impulsividad I-7 (Eysenck, Pearson, Easting, y Allsopp, 1985). Este cuestionario de 54 ítems de formato verdadero-falso está diseñado para medir impulsividad y atrevimiento. Eysenck et al. (1985) definen la impulsividad como el acto de comportarse sin pensar y ser consciente del riesgo implicado en una conducta determinada. El atrevimiento se conceptualiza como el hecho de realizar una conducta a pesar de ser consciente del riesgo que éste conlleva. La subescala de impulsividad está formada por 19 ítems, y la de atrevimiento por 16. Los 19 ítems restantes configuran una subescala de empatía. Eysenck et al. (1985) hallaron coeficientes de fiabilidad por encima de 0,80 para las subescalas de impulsividad y atrevimiento. La fiabilidad test-retest de estas dos subescalas es 0,78 y 0,90, respectivamente.

Otra de las escalas más utilizadas es la escala de Impulsividad Funcional y Disfuncional de Dickman D-II (1990). Consta de 46 ítems en formato verdadero-falso, de los cuales 11 corresponden a la subescala de Impulsividad Funcional y 12 a la subescala de Impulsividad Disfuncional. Los 23 ítems restantes constituyen ítems de relleno. Ambas subescalas han demostrado tener buenas propiedades psicométricas; los coeficientes de fiabilidad para las subescalas Funcional y Disfuncional son 0,74 y 0,85, respectivamente (Dickman, 1990).

Otra escala comúnmente empleada es la Escala de Conducta Impulsiva UPSS (Whiteside y Lynam, 2001), que consta de 45 ítems medidos en una escala Likerts de 4 puntos que oscilan entre 1 (*completamente de acuerdo*) a 4 (*completamente en desacuerdo*). La escala se divide en cuatro subescalas: urgencia, falta de premeditación, falta de perseverancia y búsqueda de sensaciones. Cada una de las cuatro escalas ha demostrado una alta consistencia interna, con coeficientes de fiabilidad que oscilan entre 0,77 y 0,91 (Schmidt, Gay, d'Acremont, y Van der Linden, 2008; Van der Linden et al., 2006).

Aunque los instrumentos previamente mencionados suelen ser los más empleados, existen otros cuestionarios que evalúan aspectos relacionados con el constructo de impulsividad tales como el Cuestionario de Personalidad Tridimensional (TPQ; Cloninger, Przybeck, y Svarkic, 1991), el inventario de Temperamento y Carácter (TCI; Cloninger, Przybeck, Svarkic, y Wetzel, 1994), la Escala de Búsqueda de Sensaciones (SSS) perteneciente al Cuestionario de Personalidad Zuckerman-Kuhlman (Zuckerman, Kuhlman, Joireman, Teta, y Kraft, 1993), y el Cuestionario de Personalidad Multidimensional (MPQ; Patrick, Curtin, y Tellegen, 2002).

1.2.2. Pruebas conductuales o de laboratorio

Mientras que los cuestionarios de autoinforme conceptualizan el constructo de impulsividad como un rasgo estable de la personalidad (Bickel, Jarmolowicz, Mueller, Gatchalian, y McClure, 2012), las pruebas conductuales pretenden realizar una captura momentánea de como actuaría *de hecho* un individuo ante una situación o en respuesta a un estímulo (Cyders y Coskunpinar, 2011). Las pruebas conductuales superan algunos de los inconvenientes que presentan los cuestionarios tradicionales de autoinforme, tales como la escasa validez aparente y su poca utilidad en poblaciones con bajos niveles de comprensión lectora o poca conciencia de su propia conducta o pensamientos. Por otro lado, proporcionan información objetiva y fácilmente cuantificable, además de requerir poco entrenamiento para su correcta administración (Coccaro, 2003) y de ser pruebas robustas a las tendencias de falseo.

En la actualidad existen una gran cantidad de pruebas conductuales o de laboratorio para medir el constructo de impulsividad. La Tabla 1 (páginas 8 y 9) muestra las diferentes pruebas clasificadas en función del tipo de componente que se evalúa. Utilizando este criterio, existen cuatro tipos principales de pruebas

conductuales: pruebas que miden desinhibición conductual, pruebas que miden déficit de atención, pruebas que miden impulsividad cognitiva y por último, pruebas que miden toma de decisiones impulsiva.

La desinhibición conductual se define como la incapacidad de controlar una respuesta que se ha iniciado previamente (Dalley, Everitt, y Robbins, 2011) o de suprimir una respuesta automática (prepotente) (Verdejo-García, et al., 2008). Las pruebas conductuales más comúnmente usadas para evaluar este componente son la prueba *Stop Signal Reaction Time* (SSRT), la prueba *go/no-go* o la prueba Stroop. Estudios provenientes del campo de la neuroimagen han identificado numerosas áreas cerebrales asociadas con el control inhibitorio de las conductas, lo que refleja su naturaleza heterogénea (Bickel, Jarmolowicz, Mueller, Gatchalian, et al., 2012). El hecho de detener una conducta ya iniciada ha demostrado estar estrechamente relacionado con la actividad en ciertas regiones del lóbulo frontal, tales como la corteza prefrontal dorsolateral, la corteza cingulada y el córtex frontal inferior (Aron, Robbins, y Poldrack, 2004; Duncan y Owen, 2000) y también regiones subcorticales como la ínsula y el putamen izquierdo o la corteza parietal inferior (Cai y Leung, 2011; Norman et al., 2011). Diversos estudios previos han demostrado que diferentes poblaciones con trastornos de obesidad (Maayan, Hoogendoorn, Sweat, y Convit, 2011), déficit de atención con hiperactividad (Barkley, 2006; Walshaw, Alloy, y Sabb, 2010), problemas de juego patológico (Kertzman et al., 2008; Roca et al., 2008) o dependencia de diversas sustancias (Colzato, van den Wildenberg, y Hommel, 2007; Pau, Lee, y Chan, 2002) muestran déficits de inhibición conductual.

El segundo componente frecuentemente evaluado por las pruebas conductuales o de laboratorio es el déficit atencional, definido como una disminución en la capacidad de concentrarse en un estímulo del entorno y de ignorar otros estímulos irrelevantes (Barkley, 1997). Las pruebas conductuales más empleadas para evaluar el déficit de atención son la prueba *Conners Continuous Performance Task* (CCPT) (Conners, 2000) o la prueba *Trail Making Test* (Parte A) (Bowie y Harvey, 2006). En general, se considera que la atención se rige por objetivos conductuales, que a continuación dirigen el procesamiento sensorial de los estímulos (Bickel, Jarmolowicz, Mueller, Gatchalian, et al., 2012). Los objetivos conductuales se mantienen supuestamente por patrones de actividad en el cortex prefrontal dorsolateral, que modula la actividad sensorial a través de proyecciones a la corteza parietal posterior (Cabeza y Nyberg, 2000; E. K. Miller y

Cohen, 2001). Estudios previos han observado déficits de atención entre sujetos dependientes de diversas sustancias como el alcohol (Thoma et al., 2011), las metanfetaminas (Johanson et al., 2006), la cocaína (Hester, Dixon, y Garavan, 2006; Kalapatapu et al., 2011) o el tabaco (Yakir et al., 2007). Así mismo también se observan estos déficits en individuos obesos (Cserjesi, Luminet, Poncelet, y Lenard, 2009) y en jugadores patológicos (Kertzman, et al., 2008).

El tercero de los componentes es la impulsividad cognitiva. Ésta se define como un déficit en la tendencia a recoger y evaluar información procedente del entorno antes de tomar una decisión (Clark, Roiser, Robbins, y Sahakian, 2009). Las pruebas conductuales que tradicionalmente se han empleado se han empleado para medir este componente son las pruebas *Matching Familiar Figures Task* (MFFT) (Zelniker, Parsons, Ault, y Jeffrey, 1972) e *Information Sampling Task* (Clark, et al., 2009). La investigación previa señala que los individuos con un funcionamiento pobre del lóbulo frontal (Chevalier, Metz-Lutz, y Segalowitz, 2000) y, más concretamente, de la corteza prefrontal dorsolateral (de Ruiter et al., 2009), son incapaces de tener habilidades básicas de planificación lo que les hace ser impulsivos a nivel cognitivo. Otras áreas cerebrales implicadas son la corteza parietal y el núcleo estriado (de Ruiter, et al., 2009). Estudios previos han encontrado que los individuos dependientes del tabaco (Yakir, et al., 2007), los opiáceos y/o la cocaína (Cohen, Nesci, Steinfeld, Haeri, y Galynker, 2010; Fernandez-Serrano, Perez-Garcia, Rio-Valle, y Verdejo-Garcia, 2010), así como los individuos con déficit de atención con hiperactividad (Nigg, Blaskey, Huang-Pollock, y Rappley, 2002) presentan altas tasas de impulsividad cognitiva.

El cuarto y último componente evaluado por las pruebas conductuales o de laboratorio es la toma de decisiones impulsiva, que se puede definir como preferencia por reforzadores inmediatos de menor valor en comparación con reforzadores demorados de mayor valor (Bickel, Jarmolowicz, Mueller, Gatchalian, et al., 2012; L. Green y Myerson, 2004). La prueba que por antonomasia se ha utilizado para evaluar la toma de decisiones impulsiva es el *Delay Discounting* o descuento por demora, tarea objeto de estudio de la presente Tesis Doctoral. Una gran cantidad de regiones cerebrales parecen estar asociadas con la toma de decisiones impulsiva. La literatura previa ha mostrado que las zonas límbica y paralímbica del cerebro (el estriado ventral, el córtex medial orbitofrontal, el córtex cingulado posterior y el hipocampo posterior derecho) tienen una elevada activación cuando los individuos prefieren reforzadores

inmediatos de menor valor en detrimento de los demorados de mayor valor. En contraposición, cuando los individuos escogen los reforzadores demorados de mayor valor las áreas con mayor activación son las prefrontales (córtex intraparietal izquierdo, corteza prefrontal dorsolateral derecha, corteza prefrontal ventrolateral derecha, y corteza orbitofrontal derecha) (Bickel, Pitcock, Yi, y Angtuaco, 2009; Kable y Glimcher, 2010; McClure, Ericson, Laibson, Loewenstein, y Cohen, 2007; McClure, Laibson, Loewenstein, y Cohen, 2004). Estudios previos han observado que los individuos con sintomatología depresiva (Imhoff, Harris, Weiser, y Reynolds, 2014; Yoon et al., 2007), tendencias suicidas (Dombrovski et al., 2011), trastornos de alimentación (Manwaring, Green, Myerson, Strube, y Wilfley, 2011), esquizofrenia (Weller et al., 2014) o dependencia de diversas sustancias (Heil, Johnson, Higgins, y Bickel, 2006; Johnson, Bickel, y Baker, 2007; Monterosso et al., 2007; Petry, 2001) muestran una toma de decisiones impulsiva.

Tabla 1. Tareas conductuales y de laboratorio en función del componente de impulsividad evaluado

Desinhibición conductual		
<i>Stop Signal Reaction Time Task</i>	Una señal “ir” se presenta en cada uno de los ensayos, provocando que la respuesta “ir” se vuelva dominante (prepotente). La señal de “parar” sucede a la “ir” en una pequeña proporción de ensayos (ensayos de “parar”). Se ha de responder a las señales “ir” y parar la respuesta “ir” lo más rápido posible o emitir una respuesta de “parar” diferente cuando la señal “parar” aparece. La capacidad de inhibir una respuesta es generalmente mayor con latencias cortas entre los ensayos “ir” y “parar”. El procedimiento ajusta el intervalo entre ensayos a un valor en se inhiba la respuesta satisfactoriamente un 50% de los ensayos “parar”, lo que constituye su tiempo de reacción para la señal “parar” (variable dependiente).	Se presentan en cada uno de los ensayos, provocando que la respuesta “ir” se vuelva dominante (estímulos “ir”) y no reforzado cuando responde a otros (estímulos “no ir”). Generalmente, los estímulos “ir” se presentan en mayor proporción que los “no ir” favoreciendo que la respuesta “ir” sea la prepotente. La variable dependiente son los errores de comisión (falsas alarmas), los errores de omisión y el tiempo de reacción.
<i>Go/no-go Test</i>	La tarea implica diferentes condiciones en las que al individuo se le pide que conteste tan rápido como pueda a estímulos presentados en serie. Las variables dependientes son extraídas de cada condición y se comparan entre las condiciones. En algunas condiciones, es imposible que se dé una respuesta correcta (por ejemplo, decir el color de una forma geométrica). En otras, el estímulo tiene características que interfieren cognitivamente con la respuesta correcta. La respuesta correcta de esta condición consiste decir el color en el que están escritos ciertos estímulos. Sin embargo, los estímulos son palabras que se refieren a colores y las palabras están impresas en otros colores que son incongruentes con el significado semántico de esa palabra.	Al individuo se le pide que presionen una tecla del teclado lo más rápido posible ante la presentación en el ordenador de un símbolo, que puede aparecer después de intervalos interestimulares de diversa duración. La variable dependiente es la proporción de tiempos de reacción inusualmente largos.
<i>Stroop Task</i>	Se le presenta al individuo una hoja con números del 1 al 25 posicionados aleatoriamente. El individuo tiene que conectar los números en orden ascendente dibujando una línea entre ellos lo más rápidamente posible y de la forma más precisa.	Se le presenta al individuo una hoja con números del 1 al 25 posicionados aleatoriamente. El individuo tiene que conectar los números en orden ascendente dibujando una línea entre ellos lo más rápidamente posible y de la forma más precisa.
Déficit de atención		
<i>Continuos Performance Task (CPT)</i>	Esta tarea en formato computerizado muestra letras separadas por un breve intervalo interestimular. El individuo tiene que pulsar una tecla del teclado o el ratón del ordenador cuando una letra objetivo previamente identificada aparezca en la pantalla. En algunas versiones de la tarea, el objetivo se define como “cualquier letra que no sea la X. En otras, el objetivo es la aparición de una secuencia de dos letras (por ejemplo, X seguido de A). Las variables dependientes son el tiempo de reacción y los errores de omisión y comisión.	Al individuo se le pide que presionen una tecla del teclado lo más rápido posible ante la presentación en el ordenador de un símbolo, que puede aparecer después de intervalos interestimulares de diversa duración. La variable dependiente es la proporción de tiempos de reacción inusualmente largos.
<i>Simple Reaction Test</i>	Al individuo se le pide que presionen una tecla del teclado lo más rápido posible ante la presentación en el ordenador de un símbolo, que puede aparecer después de intervalos interestimulares de diversa duración. La variable dependiente es la proporción de tiempos de reacción inusualmente largos.	Se le presenta al individuo una hoja con números del 1 al 25 posicionados aleatoriamente. El individuo tiene que conectar los números en orden ascendente dibujando una línea entre ellos lo más rápidamente posible y de la forma más precisa.
<i>Trail Making Test</i>		

<i>Digit Span Tasks</i>	En cada ensayo se presentan una secuencia de símbolos (letras o números individuales en formato auditivo o visual. Al individuo se le pide que repita la secuencia en el orden presentado. El número de símbolos se va incrementando a través de los ensayos. La variable dependiente es la longitud máxima de la secuencia que el individuo puede repetir sin cometer errores en un 50% de los ensayos.
<i>Matching Familiar Figures Test (MMFT)</i>	<p>Impulsividad cognitiva</p> <p>Al individuo se le presentan diversas plantillas con un dibujo estándar de un ítem familiar (por ejemplo, una bicicleta) y otros seis dibujos de los cuales cinco son similares al dibujo estándar y uno es idéntico. El individuo tiene que identifique el dibujo idéntico al estándar. Los errores cometidos son comunicados al individuo, que se le permite continuar con la misma plantilla.</p>
<i>Information Sampling Task</i>	<p>Al individuo se le presenta una cuadrícula de cajas grises de 5x5 en la pantalla del ordenador. El color gris de cada caja esconde uno de dos colores que puede ser descubierto haciendo click sobre la caja. La tarea consiste en decidir cuál es el color que está en la mayoría de las cajas. El individuo puede abrir tantas cajas como quiera antes de tomar una decisión. La variable dependiente es el número de cajas abiertas antes de tomar la decisión.</p> <p>Toma de decisiones impulsiva</p>
<i>Gambling Tasks</i>	<p>Estas tareas miden los efectos de la toma de riesgos. Por ejemplo, el Iowa Gambling Task presenta cuatro montones de cartas en la pantalla del ordenador. A los sujetos se les dice que pueden levantar una carta de un montón que elijan en cada turno, y que reciben dinero cada vez que levantan una carta. Sin embargo, algunas de las cartas también resultan en una pérdida de dinero. Cada uno de cuatro los montones tiene una distribución diferente de ganancias y pérdidas, de tal forma que dos de los montones son ventajosos para el sujetos y otros dos desventajosos.</p>
<i>Delay Discounting Task</i>	<p>Esta tarea obtiene el valor subjetivo de un reforzador de mayor valor cuya entrega es demorada en comparación con un reforzador de menor valor que se obtiene inmediatamente. El individuo tiene que escoger entre un reforzador (generalmente dinero) inmediato de menor valor o un reforzador demorado de menor valor. El reforzador inmediato de menor valor se va ajustando a través de los ensayos hasta detectar la cantidad más pequeña del mismo con la que el sujeto se mostraría indiferente entre escoger cualquiera de los reforzadores (esto es, el punto de indiferencia). La variable dependiente es tasa de descuento por demora (k), que es la velocidad a la pierde su valor el reforzador inmediato.</p>

1.2.3. Comparación de los cuestionarios de auto informe con las pruebas conductuales o de laboratorio

A pesar de que algunos estudios muestran que la ejecución en diferentes pruebas conductuales o de laboratorio correlaciona con cuestionarios de autoinforme que miden impulsividad o constructos relacionados (búsqueda de sensaciones, extraversion o propensión al riesgo) (Kirby, Petry, y Bickel, 1999; Richards, Zhang, Mitchell, y de Wit, 1999; Swann, et al., 2002), lo cierto es que hay un consenso creciente acerca de la escasa correlación entre ambos tipos de pruebas (Clark, Robbins, Ersche, y Sahakian, 2006; Crean, de Wit, y Richards, 2000; Cyders y Coskunpinar, 2011, 2012; Jacob et al., 2010; Lane, Cherek, Rhodes, Pietras, y Tcheremissine, 2003; S. H. Mitchell, 1999; Reynolds, Ortengren, et al., 2006; Reynolds, Richards, Horn, y Karraker, 2004; Stahl et al., 2014). Este resultado evidencia de nuevo la naturaleza multifactorial del constructo de impulsividad (Kirby y Finch, 2010) y sugiere que estas pruebas están midiendo diferentes componentes subyacentes a la conducta impulsiva (Cyders y Coskunpinar, 2012). La pobre relación entre ambos tipos de pruebas puede explicarse por sus diferencias en varios aspectos fundamentales: en las pruebas de autoinforme, los participantes han de reconocer y referir sus propias tendencias conductuales en varios contextos y en comparación con otros individuos, y estas auto-percepciones no siempre reflejan la conducta real de forma precisa. Por el contrario, la ejecución en las pruebas conductuales es objetiva y menos sensible a una auto-percepción sesgada. Por otro lado, las pruebas conductuales suelen medir una dimensión específica de la conducta impulsiva en un momento determinado, lo que puede limitar su generalización a contextos más amplios. En cambio, las pruebas de autoinforme están diseñadas para medir tendencias generales o rasgos estables (Cyders y Coskunpinar, 2011; Reynolds, Ortengren, et al., 2006).

1.3. Relación entre la impulsividad y el consumo de drogas

La conducta impulsiva parece constituir tanto una causa como una consecuencia de la conducta de consumo de drogas (de Wit, 2009). Por un lado, la impulsividad constituye un factor de riesgo para la experimentación con las drogas, su consumo problemático y la incapacidad de abstenerse de las mismas (Jentsch y Pennington, 2014; Loree, Lundahl, y Ledgerwood, en prensa; Tarter, Kirisci, Feske, y Vanyukov, 2007).

Estudios previos señalan también que el uso de drogas puede en sí mismo incrementar la conducta impulsiva, tanto a través de los efectos directos de su consumo agudo (Field, Wiers, Christiansen, Fillmore, y Verster, 2010) como por los efectos de su uso prolongado (Fillmore y Rush, 2002). A continuación se expone un resumen de la literatura previa que apoya la relación entre la conducta impulsiva y el uso de drogas.

1.3.1. La impulsividad como factor de riesgo para el inicio del uso de drogas, la transición del inicio al uso regular, el posterior desarrollo de trastornos y los resultados de tratamiento

Estudios transversales y longitudinales demuestran que los altos niveles de impulsividad temprana aumentan las probabilidades de experimentar con drogas, usarlas de forma regular y desarrollar trastornos debido a su uso. Aunque este tipo de estudios emplean diferentes cuestionarios de autoinforme y pruebas conductuales, todos apoyan la noción de la impulsividad como un rasgo de personalidad que incrementa el riesgo para el inicio del uso de drogas (de Wit, 2009).

Por un lado, diferentes medidas de impulsividad han demostrado predecir la transición del inicio de consumo de drogas a su uso regular y persistente (Balevich, Wein, y Flory, 2013; Chase y Hogarth, 2011). En este sentido, el estudio de neuroimagen de Norman, et al., (2011) demostró que el déficit en la activación de la corteza frontal, motora bilateral, cingulada así como del putamen izquierdo y lóbulo temporal medial durante la ejecución de pruebas que miden inhibición conductual predijo la transición del consumo aislado de sustancias a su consumo problemático.

Por otro lado, estudios previos demuestran que los altos niveles de impulsividad evaluada tanto con pruebas de autoinforme como con pruebas de laboratorio en poblaciones de niños (Tarter, et al., 2007; Tarter, Kirisci, Habeych, Reynolds, y Vanyukov, 2004) y adultos jóvenes (Sher, Bartholow, y Wood, 2000) predicen el desarrollo posterior de trastornos por consumo de sustancias y la edad de aparición de los mismos (Tarter et al., 2003). Estos resultados se confirman con otros estudios que emplearon medidas fisiológicas asociadas a la desinhibición de respuesta como potenciales evocados (Iacono y McGue, 2006) o los movimientos oculares (Habeych, Folan, Luna, y Tarter, 2006).

Así mismo, la impulsividad ha demostrado ser un predictor robusto de los resultados de tratamiento para el abuso de diferentes drogas. Estudios realizados con poblaciones de fumadores que emplean cuestionarios de autoinforme (Doran, Spring, McChargue, Pergadia, y Richmond, 2004; Helstrom, Hutchison, y Bryan, 2007; Nieve et al., 2011), pruebas de laboratorio (Dallery y Raiff, 2007; Yoon, et al., 2007) o una combinación de ambas (Krishnan-Sarin et al., 2007; Powell, Dawkins, West, Powell, y Pickering, 2010; Sheffer et al., 2012) han encontrado que los altos niveles de impulsividad están asociados con una menor probabilidad de encontrarse abstinentes tras el tratamiento y con mayores tasas de recaída. Estos resultados han sido replicados en poblaciones dependientes de otras sustancias como alcohol (Charney, Zikos, y Gill, 2010; Evren, Durkaya, Evren, Dalbudak, y Cetin, 2012; Joos et al., 2013), cannabis (Carpenter, Schreiber, Church, y McDowell, 2006; Stanger et al., 2012), cocaína (Moeller, et al., 2001; Patkar et al., 2004) y opiáceos (Poirier et al., 2004; Roll, Saules, Chudzynski, y Sodano, 2004).

Los resultados de los estudios anteriormente mencionados en este apartado han recibido apoyo empírico por parte de la investigación en el ámbito animal. A este respecto, se ha observado mediante diferentes pruebas de laboratorio que la toma de decisiones impulsiva predice mayores tasas de auto administración de cocaína (Anker, Perry, Gliddon, y Carroll, 2009; Dalley et al., 2007; Perry, Larson, German, Madden, y Carroll, 2005; Perry, Nelson, y Carroll, 2008), alcohol (Poulos, Le, y Parker, 1995) y metilfenidato (Marusich y Bardo, 2009) en ratas.

1.3.2. La impulsividad como consecuencia del uso de drogas: efectos agudos del consumo y de la abstinencia

De forma complementaria al punto anterior, el consumo de sustancias también puede afectar a la conducta impulsiva. Algunos estudios experimentales demuestran que la administración de drogas de forma aguda tiene efectos sobre la impulsividad, que varía en función de ciertos parámetros como el tipo de sustancia, la dosis administrada y los participantes del estudio (de Wit, 2009). Un factor que influye en los resultados encontrados es el tipo de evaluación que se ha empleado para medir el constructo de impulsividad. Por ejemplo, de Wit, Crean, y Richards (2000) observaron que la administración aguda de alcohol afecta de forma negativa al control inhibitorio evaluado a través de la tarea *go/no-go*. Este hallazgo fue replicado en el estudio posterior de M. A. Miller y Fillmore (2014). Sin embargo, otros estudios demuestran

que la ingesta aguda de dosis moderadas de alcohol (equivalentes a 3-4 bebidas alcohólicas) no afecta al descuento por demora de adultos jóvenes (Ortner, MacDonald, y Olmstead, 2003; Richards, et al., 1999). No obstante, un estudio encontró incrementos de las tasas de descuento por demora tras las ingesta aguda de alcohol en bebedores sociales (Reynolds, Richards, y de Wit, 2006). Los resultados observados cuando se administran otras drogas como la d-anfetamina parecen ser más consistentes. Esta sustancia parece provocar reducciones de la conducta impulsiva tanto en estudios llevados a cabo con la tarea de descuento por demora como en otros que emplean medidas de desinhibición conductual (de Wit, et al., 2000; de Wit, Enggasser, y Richards, 2002). Sin embargo, otros estudios en los que se administran sustancias como tetrahidrocannabinol (McDonald, Schleifer, Richards, y de Wit, 2003), diazepam (Acheson, Richards, Reynolds, y de Wit, 2006) o naltrexona (J. M. Mitchell, Tavares, Fields, D'Esposito, y Boettiger, 2007) no observan ningún efecto significativo en las tasas de descuento por demora.

A pesar de la falta de consenso relativa a los efectos de la administración aguda de diferentes sustancias sobre diversas medidas de impulsividad, la investigación previa acerca de los efectos de la abstinencia o retirada aguda de drogas sobre la impulsividad parece ser más consistente. La impulsividad medida a través de tareas de laboratorio que miden toma de decisiones impulsiva o desinhibición conductual parece incrementarse tras un periodo agudo de deprivación de nicotina (Ashare y Hawk, 2012; Dawkins, Powell, West, Powell, y Pickering, 2007; Field, Santarcangelo, Sumnall, Goudie, y Cole, 2006; Yi y Landes, 2012) u opiáceos (Giordano et al., 2002). Resultados similares se han observado con modelos animales: la impulsividad aumenta tras la retirada aguda de cocaína (Winstanley et al., 2009) en ratas y de PCP en monos rhesus (Carroll, Mach, La Nasa, y Newman, 2009).

1.4. El descuento por demora (*delay discounting*) como medida conductual de impulsividad

1.4.1. Definición del descuento por demora

El fenómeno del descuento por demora (*delay discounting*) constituye un área de investigación en constante crecimiento en los últimos años con implicaciones para diversos problemas socialmente importantes como la obesidad, el consumo de drogas o el juego patológico (Odum, 2011a). El número de estudios que se han publicado sobre

este tópico ha crecido rápidamente en los últimos años (Madden y Johnson, 2010) y *Pubmed* lista 261 artículos publicados (marzo, 2015) con las palabras claves “*delay discounting*” como título.

El descuento por demora constituye un índice conductual de toma de decisiones impulsiva que proviene de la economía conductual, un campo híbrido que integra los conocimientos de la psicología y la economía (Sloan y Wang, 2008). El objetivo principal de estudio de la economía conductual es entender la naturaleza de la toma de decisiones tanto racional como irracional, por lo que esta aproximación se ha aplicado tanto a la conducta normativa como a la conducta adictiva (Kahneman y Tversky, 2000; Vuchinich y Heather, 2003). El descuento por demora es un índice cuantitativo que describe la velocidad a la que un reforzador pierde su valor a medida que aumenta el tiempo para recibirla (Bickel y Marsch, 2001; Reynolds, Ortengren, et al., 2006). También puede ser considerado como un índice de la preferencia de un individuo por un reforzador inmediato de menor valor en comparación con un reforzador demorado de mayor valor, similar a la capacidad de demorar la gratificación (MacKillop et al., 2011). Aunque este índice de descuento puede calcularse de diversas maneras (L. Green y Myerson, 2004; Mazur, 1987; Myerson, Green, y Warusawitharana, 2001; Yoon y Higgins, 2008), en todas ellas cuanto más precipitadamente pierda su valor el reforzador demorado para un individuo concreto, más impulsivo será ese individuo.

1.4.2. Los modelos del descuento por demora

Uno de los abordajes más desarrollados en relación a la formulación del descuento por demora proviene del campo de la economía. Las teorías económicas asumen que el valor del reforzador demorado se descuenta de forma exponencial: esto es, por cada unidad de tiempo que constituye la demora de la entrega de tal reforzador, el valor del mismo decrece (o es descontado) en una proporción fija (Kirby, 1997). El modelo exponencial asume un modelo racional de elección, es decir, entiende que las preferencias de los individuos permanecen constantes a lo largo del tiempo y no se revierten (Ainslie y Haslam, 1992). Este modelo asume que el descuento por demora se ajustara a la siguiente ecuación exponencial:

$$V = Ae^{-kd} \quad (1)$$

En la ecuación 1 V es el valor descontado del reforzador demorado, A es la cantidad de reforzador, d es la demora hasta su entrega y k corresponde a la tasa de descuento (e es la base del logaritmo natural, 2,718). El parámetro k sirve como valor que permite operativizar la impulsividad; a mayor valor de k mayor es la tasa de descuento por demora, y por tanto mayor es la impulsividad (Reynolds, 2006b).

Sin embargo, la inversión de las preferencias es un fenómeno típico que se da tanto en la vida diaria de las personas como en el contexto experimental. Por ejemplo, las personas se proponen hacer dieta durante cierto tiempo pero después deciden que comer una hamburguesa hoy es más valioso que la lenta pérdida de peso que supondría seguir la dieta. De la misma forma, las conductas adictivas se caracterizan por un deseo persistente de autocontrol (es decir, abstenerse del consumo de la sustancia de referencia), acompañado de continuos fracasos a la hora de lograrlo. En este sentido, el drogodependiente prefiere los beneficios a largo plazo de mantenerse abstinentemente a cambio de renunciar a un placer inmediato como es el consumo de la droga, pero la inversión de las preferencias ocurre siempre que se dispone de un acceso inmediato a la sustancia.

El modelo exponencial del descuento por demora cuenta con la limitación de no tener en cuenta la posibilidad de la inversión de las preferencias. Otra limitación importante de este modelo es que no tiene apoyo empírico de la investigación conductual. Estudios llevados a cabo tanto en humanos (Myerson y Green, 1995; Ohmura, Takahashi, Kitamura, y Wehr, 2006; Rachlin, Rainieri, y Cross, 1991; Simpson y Vuchinich, 2000) como en animales muestran desviaciones sistemáticas del modelo exponencial (Logue, 1988; Mazur, 1997), ajustándose mejor a un modelo hiperbólico. El modelo hiperbólico se define por una atenuación en la velocidad de devaluación del reforzador a medida que aumenta la demora para recibirla. Esto es, por cada unidad de tiempo que constituye la demora de la entrega de tal reforzador, el valor del mismo decrece (o es descontado) en una proporción cada vez más pequeña. Esto significa que el valor del reforzador se descuenta de forma más precipitada cuando las demoras son cortas, pero el descuento se modera a medida que la demora aumenta (Kirby, 1997). El modelo asume que el descuento por demora se ajusta a la siguiente función hiperbólica (Mazur, 1987):

$$V = A/(1+kD) \quad (2)$$

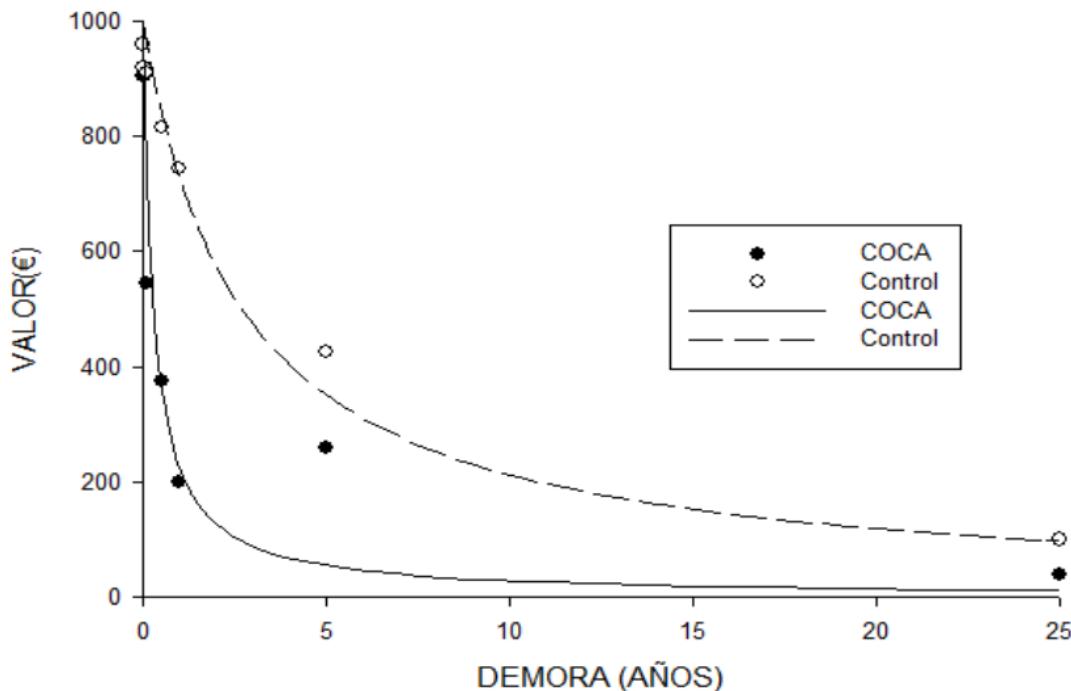
En la ecuación 2 V es el valor descontado del reforzador demorado, A es la cantidad de reforzador, d es la demora hasta su entrega y k corresponde a la tasa de descuento por demora.

El modelo hiperbólico ha demostrado ajustarse de forma precisa a la función de descuento por demora, explicando al menos un 85% de la varianza cuando se emplean agua y comida como reforzadores en animales (Richards, Mitchell, DeWit, y Seiden, 1997) y dinero tanto real como hipotético en humanos (L. Green, Fry, y Myerson, 1994; Kirby y Herrnstein, 1995; Madden, Petry, Badger, y Bickel, 1997; Myerson y Green, 1995; Rachlin, et al., 1991; Richards, et al., 1999).

Las medidas de descuento por demora se han diseñado típicamente para determinar la tasa de devaluación del reforzador demorado a lo largo del tiempo. Para ello, se suele el emplear el procedimiento de ajuste de demoras (Mazur, 1987; Rodriguez y Logue, 1988) o el procedimiento de ajuste de cantidades (S. H. Mitchell, 1999; Reynolds, 2006a; Richards, et al., 1999).

En los estudios que conforman la presente Tesis Doctoral se ha empleado el procedimiento de ajuste de cantidades. En este procedimiento, al individuo se le presentan diversas elecciones entre un reforzador demorado de mayor valor estándar (por ejemplo, 1000€) y un reforzador de menor valor disponible inmediatamente que se ajusta hasta que es percibido por el individuo como equivalente al valor del reforzador demorado (L. Green, et al., 1994). Este punto de equivalencia es el denominado punto de indiferencia para una demora particular. Cuando se han obtenido varios puntos de indiferencia correspondientes a varias demoras, entonces se puede representar una curva de indiferencia o de descuento por demora (ver Figura 1, siguiente página). La importancia de las curvas de indiferencia radica en que permiten determinar empíricamente la forma de la función y derivar también de forma empírica la velocidad a la que se descuentan los reforzadores demorados (Bickel y Marsch, 2001).

Figura 1. Curvas de indiferencia o de descuento por demora



En la Figura 1 se muestran las curvas de indiferencia de un grupo de sujetos dependientes de la cocaína y de un grupo control, ambas generadas a partir de la media geométrica de k . Los círculos muestran los puntos de indiferencia medios en una determinada demora para el grupo dependiente de cocaína (negro) y para el grupo control (blanco). El grupo de sujetos dependiente de la cocaína muestra una curva de descuento por demora significativamente más pronunciada, y, por tanto, una mayor impulsividad en comparación con el grupo de sujetos controles no dependientes de la cocaína.

De forma adicional a la ecuación (2), algunos investigadores emplean el área bajo la curva (*area under the curve; AUC*) (Myerson, et al., 2001) como método matemático para obtener la tasa de descuento por demora. Al contrario que los modelos exponencial o hiperbólico, el área bajo la curva se calcula empleando directamente los puntos de indiferencia. Así, el área bajo la curva proporciona un índice ateórico del descuento que evita errores sistemáticos de ajuste derivados de la asunción de un modelo particular (Odum, 2011b; Odum y Rainaud, 2003). La ecuación que se emplea para calcular el área bajo la curva es la siguiente:

$$(x_2 - x_1) [(y_1 + y_2) / 2] \quad (3)$$

En la ecuación 3 x_2 y x_1 representan las sucesivas demoras, mientras que y_1 e y_2 son los puntos de indiferencia asociados a esas demoras. Antes de calcular el área bajo la curva, los valores x e y se normalizan dividiendo cada valor por el valor más alto de x e y , respectivamente, proporcionando valores entre 0 (no descuento por demora) y 1 (máximo descuento por demora).

1.4.3. Procedimientos para evaluar el descuento por demora

Se han desarrollado tres tipos de herramientas de evaluación del descuento por demora en humanos: las que emplean reforzadores hipotéticos, las que usan reforzadores reales y, por último, las medidas a tiempo real. Los tres comparten la característica de tratarse de procedimientos de elección que requieren que los individuos escojan entre reforzadores disponible inmediatamente (o tras una corta demora) o reforzadores que son demorados (Reynolds, 2006b).

La mayor parte de la investigación sobre descuento por demora llevada a cabo en humanos ha empleado el procedimiento de reforzadores hipotéticos, que implica que tanto los reforzadores como las demoras son imaginarios. Los individuos tienen que hacer sucesivas elecciones entre los reforzadores inmediatos y demorados, pero no reciben ningún reforzador ni experimentan la demora asociada a el mismo. La forma prototípica en la que se redacta cada pregunta en la que el individuo tiene que realizar la elección sería la siguiente: “¿Qué preferirías, 500 € ahora o 1000 € mañana?”. En los procedimientos de evaluación basados en preguntas hipotéticas también se pueden presentar elecciones entre dos reforzadores demorados, uno disponible después de una demora corta y el otro tras una demora más larga (ver L. Green, Myerson, y Macaux, 2005). Los procedimientos de evaluación del descuento por demora que emplean reforzadores hipotéticos ofrecen las ventajas de ser económicos y de poder emplearse en cortos periodos de tiempo.

El segundo tipo de herramientas de evaluación del descuento por demora emplea reforzadores reales (ver L. Green y Myerson, 2004). Con el objetivo de incrementar la validez aparente, en este tipo de procedimiento se selecciona al azar una de las respuestas dadas por el individuo durante la evaluación para que éste reciba cierta cantidad de dinero inmediato o demorado en función de su elección. Diversos estudios llevados a cabo tanto con población general (Johnson y Bickel, 2002; Lagorio y Madden, 2005) como con población drogodependiente (Baker, Johnson, y Bickel, 2003)

no han encontrado diferencias sistemáticas en función de si los reforzadores empleados en el procedimiento de descuento por demora son hipotéticos o reales, lo que parece indicar que el empleo de estos dos tipos de reforzadores proporciona evaluaciones comparables del descuento por demora.

El tercer tipo de procedimientos de evaluación del descuento por demora consiste en las medidas a tiempo real (ver Reynolds, 2006a). Este procedimiento difiere de los dos anteriores en que los participantes experimentan todas las consecuencias derivadas de sus elecciones (tanto las demoras como los reforzadores seleccionados) en tiempo real mientras completan la evaluación (ver Lane, Cherek, Pietras, y Tcheremissine, 2003; Reynolds y Schiffbauer, 2004). Algunos estudios utilizan medidas a tiempo real emplean reforzadores primarios (ver Kirk y Logue, 1997), pero la mayoría usan reforzadores monetarios. En las medidas a tiempo real, el individuo ha de escoger entre recibir un reforzador de mayor valor demorado y probabilístico (por ejemplo, una probabilidad del 35% de recibir 0,30€) o recibir un reforzador de menor valor inmediato y seguro (por ejemplo, una probabilidad del 100% de recibir 0,15€). Las medidas a tiempo real implican demoras más cortas (inferiores a 90 segundos) y cantidades de dinero más pequeñas (inferiores a 0,5€) que los procedimientos que emplean reforzadores hipotéticos y reales. Las medidas a tiempo real cuentan con la ventaja de ser una herramienta más sensible para determinar variaciones a corto plazo en las tasas de descuento por demora cuando se inducen experimentalmente cambios en los individuos (por ejemplo, cuando se administran drogas de forma aguda) porque los individuos experimentan las consecuencias de sus elecciones mientras se encuentran en tal estado inducido (ver McDonald, et al., 2003; Reynolds, Richards, Dassinger, y De Wit, 2004). Así mismo, las medidas a tiempo real pueden ser útiles cuando los sujetos experimentales son niños, porque requieren menor grado de abstracción a la hora de evaluar los reforzadores y las demoras que cuando se emplean reforzadores hipotéticos o reales (Reynolds, 2006b). Sin embargo, las medidas a tiempo real suponen un mayor coste y tiempo de administración. Además, algunos autores también cuestionan que estas medidas a tiempo real evalúen el descuento por demora realmente ya que no aislan la demora como el único factor influyente en la elección, al también modificar la probabilidad de recibir los diferentes reforzadores (Lagorio y Madden, 2005; Madden et al., 2004). Algunos estudios han comparado los resultados producidos por las medidas a tiempo real con aquellos hallados cuando se emplean reforzadores reales o hipotéticos,

encontrando correlaciones positivas significativas por encima de 0,50, lo que sugiere una asociación moderada entre ambos procedimientos (Lane, Cherek, Pietras, et al., 2003; Reynolds, 2006a).

1.5. El descuento por demora y la dependencia de sustancias

El descuento por demora constituye un fenómeno fundamental a la hora de explicar la drogodependencia (Bickel y Johnson, 2003). Las altas tasas de descuento por demora comunes entre los drogodependientes tienen una relevancia sustancial para entender la conducta adictiva, ya que permiten explicar la preferencia individual por los efectos transitorios asociados al consumo de drogas a costa de los beneficios futuros que supone la abstinencia de las mismas (MacKillop, et al., 2011). Además, el excesivo descuento por demora posibilita la comprensión del fallo de autocontrol, otra característica distintiva de la conducta adictiva y un síntoma clínico del trastorno por uso de sustancias (American Psychiatric Association, 2013; Lyvers, 2000). Así mismo, el descuento por demora permite entender el fenómeno de ambivalencia relativa a la abstinencia que con frecuencia se observa en la práctica clínica. Aunque la inversión de las preferencias puede estar presente en cualquier conducta humana, la conducta adictiva se caracteriza especialmente por la vacilación continua entre las inclinaciones por la abstinencia y las inclinaciones por la continuación del consumo (Ainslie, 2001).

La relación entre el descuento por demora y la drogodependencia ha sido bastante estudiada en las dos últimas décadas, aunque hay algunos aspectos a los que se ha prestado escasa atención (Bickel, Koffarnus, Moody, y Wilson, 2014). A continuación se realizará una revisión de los principales tópicos de estudio previo que permiten señalar al descuento por demora como marcador conductual de la conducta adictiva, para luego mencionar las limitaciones de estos estudios que fundamentan los objetivos de la presente Tesis Doctoral.

1.5.1. El descuento por demora permite diferenciar a los individuos drogodependientes de los controles

La investigación previa que compara las tasas de descuento por demora de diferentes poblaciones de drogodependientes con sujetos controles no dependientes ha mostrado de forma reiterada que los sujetos dependientes de sustancias presentan tasas de descuento por demora significativamente mayores. El primer artículo publicado a

este respecto fue realizado con un grupo de sujetos dependientes de opiáceos (Madden, et al., 1997). En este estudio se observó que el grupo dependiente de opiáceos descontó el valor del dinero demorado más que el grupo control. En concreto, para éste último grupo los 1000\$ demorados perdieron la mitad de su valor a los 37 meses, mientras que esta devaluación ocurrió a los 4,5 meses en el grupo dependiente de opiáceos. Este hallazgo se replicó en posteriores estudios que también emplearon participantes dependientes de opiáceos (Kirby y Petry, 2004; Kirby, et al., 1999; Madden, Bickel, y Jacobs, 1999), y se generalizó a otras poblaciones dependientes de otras sustancias, como alcohol (Bjork, Hommer, Grant, y Danube, 2004; Bobova, Finn, Rickert, y Lucas, 2009; J. M. Mitchell, Fields, D'Esposito, y Boettiger, 2005; Petry, 2001), tabaco (Baker, et al., 2003; Bickel, Odum, y Madden, 1999; Bickel, Yi, Kowal, y Gatchalian, 2008; Johnson, et al., 2007; S. H. Mitchell, 1999; Odum, Madden, y Bickel, 2002; Reynolds, 2004; Reynolds, Leraas, Collins, y Melanko, 2009; Rezvanfard, Ekhtiari, Mokri, Djavid, y Kaviani, 2010), cocaína (Bickel et al., 2011; Camchong et al., 2011; Coffey, Gudleski, Saladin, y Brady, 2003; Heil, et al., 2006; Kirby y Petry, 2004) y metanfetaminas (Hoffman et al., 2006; Monterosso, et al., 2007). El cannabis supone una excepción a estos estudios, ya que aunque se observa una tendencia a un mayor descuento por demora por parte de los sujetos dependientes de cannabis en comparación con los controles, el tamaño del efecto de esta comparación es más pequeño que el encontrado en estudios realizados con otras poblaciones de drogodependientes (Johnson et al., 2010). En conclusión, y a pesar de esta excepción mencionada y de la variedad de métodos empleados en relación a las técnicas de muestreo y de reforzadores empleados en la evaluación del descuento por demora, los estudios previos muestran de forma consistente que los sujetos drogodependientes presentan mayores tasas de descuento por demora en comparación con sujetos controles.

1.5.2. El descuento por demora constituye un predictor del consumo de drogas

En comparación con el volumen de publicaciones relativas al punto anterior, pocos estudios han evaluado si el descuento constituye un predictor robusto del inicio del consumo de drogas o de la dependencia a las mismas. El principal motivo de la escasez de estudios sobre este tópico se debe a que la única forma de evaluarlo de forma fiable y precisa es mediante estudios longitudinales que midan el descuento por demora antes de que se produzca cualquier consumo de drogas y realicen seguimientos para

comprobar qué individuos continúan abstinentes o han consumido/consumen una droga determinada.

Ayduk et al. (2000) realizaron el primer estudio relativo a este tópico empleando una tarea similar a la de descuento por demora denominada demora de la gratificación. Los autores encontraron que la demora de la gratificación evaluada en niños preescolares predijo el consumo de cocaína o crack durante la adultez. Un estudio posterior también observó una relación positiva entre el descuento por demora evaluado en la línea base y el inicio posterior del consumo de tabaco en una muestra de adolescentes evaluados en tres olas (Audrain-McGovern et al., 2009). Así mismo, el descuento por demora constituyó un predictor significativo del posterior aumento del consumo de tabaco: un incremento de una desviación típica en el descuento por demora de la línea base supuso un incremento de un 11% de probabilidades de aumentar el consumo de cigarrillos.

La falta de datos adicionales que confirmen los hallazgos de estos dos estudios limita la capacidad predictiva del descuento por demora sobre el consumo posterior de sustancias. No obstante, es importante señalar que el descuento por demora también ha demostrado ser un predictor robusto de la auto administración de drogas en investigaciones realizadas con animales (Anker, et al., 2009; Diergaarde et al., 2008; Perry, et al., 2008).

1.5.3. La relación entre el descuento por demora y la gravedad de la adicción

El descuento por demora ha demostrado estar asociado con diferentes parámetros que indican gravedad de la adicción. Estudios previos demuestran de forma consistente que las tasas de descuento por demora correlacionan positivamente con el número de cigarrillos fumados y/o la cantidad de nicotina consumida al día (Johnson, et al., 2007; MacKillop y Kahler, 2009; Ohmura, Takahashi, y Kitamura, 2005; Reynolds, 2004), la cantidad máxima de cocaína consumida (Albein-Urios, Miguel Martínez-González, Lozano, Clark, y Verdejo-García, 2012), el número de años de abuso de heroína (Cheng, Lu, Han, Gonzalez-Vallejo, y Sui, 2012) y la cantidad de alcohol consumida por semana (MacKillop et al., 2010).

1.5.4. Los efectos de la abstinencia aguda en el descuento por demora

Algunos estudios previos han evaluado el efecto de un periodo agudo de abstinencia inducido experimentalmente sobre el descuento por demora. El primer estudio relativo a este tópico comparó las tasas de descuento por demora de un grupo de sujetos dependientes de opiáceos que llevaban cinco días deprivados de la droga con otro grupo de sujetos saciados de la misma (Giordano, et al., 2002). Los resultados indicaron que los sujetos en la condición de deprivación descontaron significativamente más por demora que aquellos en la condición de saciación. El aumento de las tasas de descuento por demora como consecuencia de la abstinencia aguda constituye un hallazgo que ha sido replicado en diversos estudios realizados con fumadores (Ashare y Hawk, 2012; Field, et al., 2006; S. H. Mitchell, 2004; Yi y Landes, 2012). No obstante, es conveniente señalar que aunque la abstinencia aguda afecta al descuento por demora, factores como el tipo y la magnitud del reforzador empleado en la tarea de descuento por demora parecen modular tal efecto (Bickel, et al., 2014)

1.5.5. El descuento por demora en individuos consumidores de sustancias frente a ex consumidores

Los resultados encontrados en relación a este tópico varían en función del tipo de población drogodependiente con la que se haya realizado el estudio. Por ejemplo, los ex fumadores presentan tasas de descuento por demora similares a los individuos que nunca han fumado, y ambos grupos tienen un descuento por demora inferior a los fumadores actuales (Bickel, et al., 1999; Sweitzer, Donny, Dierker, Flory, y Manuck, 2008). Este hallazgo también se ha replicado en sujetos dependientes de heroína y anfetaminas (Bretteville-Jensen, 1999). Sin embargo, estudios previos señalan que los sujetos dependientes de cocaína que dejaron de consumir 14 (Kirby y Petry, 2004) o 30 días (Heil, et al., 2006) antes de la evaluación presentan tasas de descuento por demora similares a los sujetos que continúan consumiendo. Los resultados encontrados en la población dependiente de alcohol son mixtos: los sujetos que llevan sin consumir 14 días previos a la evaluación presentan tasas de descuento similares a los sujetos que continúan consumiendo (Kirby y Petry, 2004), mientras que éstas son significativamente inferiores cuando llevan 30 días sin consumir (Petry, 2001). La inconsistencia de los resultados encontrados en función del tipo de población analizada

puede estar relacionada con la variación en los criterios relativos a la cantidad de días de abstinencia necesarios para categorizar a los sujetos como abstinentes en el momento de la evaluación (Heil, et al., 2006).

1.5.6. El descuento por demora como predictor de la abstinencia

Los hallazgos previos relativos a este tópico se han obtenido mediante la realización de estudios de laboratorio y de ensayos clínicos. En relación al primer tipo de estudios, algunos resultados han mostrado que el descuento por demora es un predictor significativo de la conducta de fumar en fumadores sin intención de dejarlo que participan en modelos de laboratorio de reforzamiento de la abstinencia (Dallery y Raiff, 2007; Mueller et al., 2009). Por otra parte, varios ensayos clínicos llevados a cabo con adolescentes fumadores de tabaco (Krishnan-Sarin, et al., 2007) y cannabis (Stanger, et al., 2012) mostraron que el descuento por demora predijo la abstinencia tras el tratamiento. Este hallazgo se ha replicado en poblaciones adultas dependientes de tabaco y/o alcohol (MacKillop y Kahler, 2009; Sheffer et al., 2014; Sheffer, et al., 2012; Yoon, et al., 2007) y de cocaína (Washio et al., 2011). No obstante, un estudio llevado a cabo con sujetos dependientes de opiáceos observó que el descuento por demora de aquellos que estaban abstinentes tras el tratamiento no difirió del de aquellos que continuaban consumiendo (Passetti, Clark, Mehta, Joyce, y King, 2008). En general, y a pesar del resultado del último estudio mencionado, los resultados previos indican que las tasas de descuento por demora se relacionan de forma sistemática con los resultados de los tratamientos de drogodependencias.

1.5.7. Cambios en el descuento por demora tras recibir tratamientos eficaces

Las tasas de descuento por demora han demostrado ser estables en períodos que oscilan entre 1 semana y 1 año cuando no se realiza ninguna intervención entre las evaluaciones (Beck y Triplett, 2009; Kirby, 2009; Simpson y Vuchinich, 2000; Takahashi, Furukawa, Miyakawa, Maesato, y Higuchi, 2007). Sin embargo, estudios previos sugieren que las tasas de descuento por demora pueden modificarse tras recibir intervenciones eficaces. A este respecto, el descuento por demora se redujo tras un entrenamiento en memoria de trabajo en sujetos dependientes de estimulantes (Bickel, Yi, Landes, Hill, y Baxter, 2011) y después de un tratamiento para la gestión adecuada de dinero en sujetos dependientes de cocaína y/o alcohol (Black y Rosen, 2011).

Existen tres estudios que realizan intervenciones basadas en técnicas de Manejo de Contingencias de forma aislada (Yi et al., 2008) o combinada con otros tratamientos (Landes, Christensen, y Bickel, 2012; Peters, Petry, LaPaglia, Reynolds, y Carroll, 2013). Dos de estas investigaciones encontraron que el MC produjo reducciones significativas del descuento por demora en fumadores (Yi, et al., 2008) y dependientes de opiáceos (Landes, et al., 2012), mientras que el tercer estudió mostró que el descuento por demora permanecía estable tras la intervención en sujetos dependientes de marihuana (Peters, et al., 2013). En resumen, la mayor parte de las investigaciones previas sugieren que el descuento por demora se reduce tras recibir intervenciones eficaces dirigidas tanto al abandono del consumo de una sustancia como a otros aspectos. Sin embargo, la escasez de estudios relativos a este tópico evidencia la necesidad de estudios adicionales que confirmen este hallazgo.

1.6. Limitaciones de la investigación previa

La investigación previa sobre la asociación entre el descuento por demora y la dependencia de sustancias presenta ciertas limitaciones. Algunas de estas limitaciones han fundamentado los objetivos de la presente Tesis Doctoral que se expondrán en el siguiente apartado de este trabajo.

Una de las limitaciones de la investigación se encuentra relacionada con el tópico relativo al descuento por demora como elemento diferenciador de los sujetos drogodependientes y de los controles. Aunque la investigación previa demuestra de forma consistente que los sujetos dependientes de diferentes sustancias tienen tasas de descuento por demora mayores que los sujetos controles no dependientes, la relación entre el descuento por demora y el policonsumo de sustancias ha sido muy poco estudiada. A este respecto, tan solo un estudio llevado a cabo con fumadores con y sin un trastorno comórbido por consumo de sustancias y con no fumadores con y sin tal trastorno mostró que los fumadores tenían tasas de descuento por demora similares a los sujetos que presentaban trastornos por consumo de sustancias (Businelle, McVay, Kendzor, y Copeland, 2010). Además, se observó que el policonsumo de sustancias no afectó a las tasas de descuento por demora, de tal forma que los sujetos dependientes de varias drogas presentaron tasas de descuento similares a aquellos dependientes de solo una sustancia. No obstante, es importante destacar que el valor de los resultados encontrados se encuentra limitado por la heterogeneidad de los sujetos

policonsumidores en términos del tipo y el número de trastornos por consumo de sustancias presentado. De hecho, en este estudio un 78,2% de los participantes asignados a cualquiera de los grupos de trastorno por consumo de sustancias, presentaban más de un trastorno de este tipo pero no se especifica cuál o cuáles eran éstos. El hecho de no controlar de forma específica el número y el tipo de sustancias consumidas por los participantes de este estudio hacen necesarios nuevos estudios con el fin de clarificar la relación entre el policonsumo de sustancias (incluyendo el tabaco) y el descuento por demora.

Otra de las grandes limitaciones de la investigación previa tiene que ver con la naturaleza transversal de la mayor parte de los estudios, que imposibilita el abordaje preciso de la relación causa-efecto entre consumo de sustancias (y la drogodependencia) y las tasas de descuento por demora. Mientras que algunos de los estudios previos sugieren que las altas tasas de descuento por demora pueden ser un factor de riesgo para el desarrollo de conductas adictivas, otros sugieren que las altas tasas de descuento por demora pueden ser una consecuencia de la adicción en sí misma (Reynolds, 2006b). Los estudios de tipo transversal realizados con sujetos dependientes y abstinentes de sustancias no permiten esclarecer la etiología relativa a los altos niveles de impulsividad que presenta la población con trastornos por consumo de sustancias (Verdejo-García, et al., 2008). La escasez de estudios longitudinales que analicen los cambios en el descuento por demora en función del estatus de consumo o abstinencia de sustancias limita los hallazgos de las investigaciones previas (Bickel, et al., 2014), y constata la necesidad de nuevos estudios que analicen las modificaciones en el descuento por demora de forma longitudinal.

Además, son muy escasos los estudios sobre descuento por demora en población dependiente de sustancias con un diagnóstico o sintomatología psiquiátrica asociada. Algunos estudios han observado que los sujetos dependientes de tabaco (Fields, Collins, Leraas, y Reynolds, 2009), alcohol (Dom, D'Haene, Hulstijn, y Sabbe, 2006) y heroína o cocaína (Liu, Vassileva, Gonzalez, y Martin, 2012) que presentan psicopatología asociada tienen un descuento por demora más alto que sujetos dependientes de la misma sustancia sin tal psicopatología. Algunos estudios han encontrado que la conducta de fumar y la sintomatología depresiva se relacionan de forma independiente con unas mayores tasas de descuento por demora (Gatchalian, Yi, Bickel, Johnson, y Baker, 2004; Imhoff, et al., 2014). Sin embargo ningún estudio ha analizado si la abstinencia

del consumo de tabaco podría modificar las tasas de descuento por demora en población fumadora con síntomas depresivos. Además, la relación entre la sintomatología depresiva y el descuento por demora en fumadores se ha estudiado siempre de forma secundaria, sin que haya sido el objetivo principal de ninguna de las investigaciones previas. Todo ello evidenció la necesidad de un estudio adicional que analizara de forma pormenorizada la interrelación entre la sintomatología depresiva, el estatus de consumo o abstinencia del tabaco y los cambios en las tasas de descuento por demora.

Por último, una cuarta limitación tiene que ver con el análisis de la eficacia de diferentes tipos de intervenciones a la hora de reducir las tasas de descuento por demora en población dependiente de sustancias. Por un lado, la escasez de estudios previos relativos a este tópico y los resultados inconsistentes hallados limitan las conclusiones que se pueden extraer de los mismos (Bickel, et al., 2014). Por otro, ningún estudio realizado hasta la fecha ha realizado evaluaciones de seguimiento a largo plazo tras la finalización de las intervenciones, con lo que es imposible conocer si las reducciones en el descuento por demora halladas en algunos de estos estudios se mantienen a lo largo del tiempo o se desvanecen. Además, algunos de estos estudios previos no realizan un control estadístico del posible efecto interactivo del estatus consumo o abstinencia y el tipo de intervención recibido, hecho que impide saber si la causa de las reducciones en el descuento por demora es el tratamiento recibido, la abstinencia del consumo o el efecto conjunto de ambos factores. Todas estas limitaciones de la investigación revelan la necesidad de nuevos estudios que analizaran la eficacia de una intervención para reducir las tasas de descuento por demora tanto al final de dicha intervención como a lo largo de seguimientos a medio y largo plazo.

2. OBJETIVOS DE LA TESIS DOCTORAL

Teniendo en cuenta algunas de las limitaciones de la investigación previa relativas a la relación entre el descuento por demora y la drogodependencia (ver apartado 5.8), el objetivo general de la presente Tesis Doctoral es evaluar la impulsividad mediante la aplicación de la tarea de descuento por demora en sujetos dependientes de sustancias. Los objetivos específicos son los siguientes:

- 1) Comparar las tasas de descuento por demora de cuatro grupos de participantes: sujetos dependientes de cocaína y nicotina, dependientes sólo de cocaína, dependientes sólo de nicotina y sujetos controles no dependientes de ninguna sustancia.
- 2) Analizar de forma longitudinal si las tasas de descuento por demora se modifican en función del estatus de consumo del tabaco al final de un Tratamiento Cognitivo Conductual (TCC) y al año de seguimiento
- 3) Evaluar los efectos principales e interactivos de la sintomatología depresiva y los cambios en el estatus de consumo de tabaco sobre las tasas de descuento por demora en fumadores que recibieron tratamiento para dejar de fumar.
- 4) Analizar el efecto diferencial de un componente de Manejo de Contingencias añadido a un TCC para dejar de fumar, sobre las tasas de descuento por demora al final de tratamiento y a los 6 meses de seguimiento.

3. PUBLICACIONES

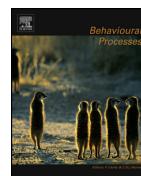
Esta Tesis Doctoral se presenta en formato de compendio de publicaciones. Para ello, se han incluido 3 estudios ya publicados en revistas internacionales con factor de impacto y un cuarto trabajo complementario que se encuentra en vías de publicación en una revista científica.

3.1. Artículo 1

Referencia: García-Rodríguez, O., Secades-Villa, R., Weidberg, S., y Yoon, J. H. (2013). A systematic assessment of delay discounting in relation to cocaine and nicotine dependence. *Behavioural Processes*, 99, 100-105. doi: 10.1016/j.beproc.2013.07.007

Resumen: Delay discounting is a measure of impulsivity describing how a reinforcer loses value as the delay to its receipt increases. Greater delay discounting is reliably observed among those with different substance use disorders (SUDs) compared to the general population. Nevertheless, the relation between delay discounting and the type and number of substances used remains unclear. The aim of this study was to compare delay discounting across four groups of participants: cocaine- and nicotine-dependent participants, cocaine-dependent only participants, nicotine-dependent only participants, and non-dependent controls. One hundred and seven participants completed a computerized delay discounting task for hypothetical monetary values. Data were fit to Mazur's hyperbolic equation to derive the discounting rate k. Results showed that delay discounting was significantly greater in the cocaine- and nicotine-dependent group, compared to the nicotine-dependent only group, compared to control group. Delay discounting was also greater in the cocaine-dependent only group relative to the nicotine-dependent only and control groups, but no differences were observed between the cocaine- and nicotine-dependent group and the cocaine-dependent only group. This study provides evidence that delay discounting differs depending on the type of SUD but not on the number of SUDs.

Factor de impacto: 1.457 (JCR 2013)



A systematic assessment of delay discounting in relation to cocaine and nicotine dependence



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ABSTRACT

Delay discounting is a measure of impulsivity describing how a reinforcer loses value as the delay to its receipt increases. Greater delay discounting is reliably observed among those with different substance use disorders (SUDs) compared to the general population. Nevertheless, the relation between delay discounting and the type and number of substances used remains unclear. The aim of this study was to compare delay discounting across four groups of participants: cocaine- and nicotine-dependent participants, cocaine-dependent only participants, nicotine-dependent only participants, and non-dependent controls. One hundred and seven participants completed a computerized delay discounting task for hypothetical monetary values. Data were fit to Mazur's hyperbolic equation to derive the discounting rate k . Results showed that delay discounting was significantly greater in the cocaine- and nicotine-dependent group, compared to the nicotine-dependent only group, compared to control group. Delay discounting was also greater in the cocaine-dependent only group relative to the nicotine-dependent only and control groups, but no differences were observed between the cocaine- and nicotine-dependent group and the cocaine-dependent only group. This study provides evidence that delay discounting differs depending on the type of SUD but not on the number of SUDs.

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1. Introduction

Delay discounting describes how a reinforcer loses value as the delay to its receipt increases. Delay discounting is an operational definition of impulsivity characterizing the tendency to choose smaller, more immediate reward over a larger, delayed one. The discounting phenomena is well-established in both animals (Freeman et al., 2009; Mazur, 2000; Reynolds et al., 2002) and human subjects (Madden and Bickel, 2010; Reynolds, 2006) with different types of reinforcers such as money (MacKillop et al., 2011), food (Epstein et al., 2009; Odum et al., 2006) or sex (Lawyer et al., 2010).

The delay discounting procedure has been used to address various socially relevant health problems behaviors (Critchfield and Kollins, 2001) but the vast majority of discounting research has been centered on substance abuse, suggesting that delay discounting is a fundamental behavioral process in substance use disorders (SUDs) (Bickel and Marsch, 2001). The delay discounting task may provide a direct assessment of the impaired decision making processes characterized for the preference for the small, relatively immediate reward (consuming a drug) over larger, more delayed

rewards (improved health, financial stability, etc.) that contribute to drug use initiation, maintenance, and relapse (Madden and Bickel, 2010). In the prototypical delay discounting task, the participant is presented with repeated choices between a relatively smaller, immediate reinforcer and a larger reinforcer available after a fixed delay. The magnitude of the smaller, immediate choice is adjusted until the two choices are deemed subjectively equal (i.e., an indifference point is reached; Rachlin et al., 1991). Indifference points are obtained for multiple delays and the pattern of these indifference points can be described mathematically using a hyperbolic discounting function (Eq. (1)) described by Mazur (1987):

$$V = \frac{A}{1 + kD}. \quad (1)$$

The equation shows how the value (V) of a reinforcer of some amount (A) is discounted as a function of delay (D) to receiving it (Mazur, 1987). The free parameter k describes the rate of discounting, with higher values of k indicating greater discounting and impulsivity. Although other models of delay discounting exist (McKerchar et al., 2009), Eq. (1) is arguably the most commonly used. For example, a recent meta-analysis of studies examining delay discounting among those with SUD noted that 70% of the studies utilized Eq. (1) (MacKillop et al., 2011).

Higher levels of delay discounting have been reliably observed among those with SUDs compared to matched controls for numerous drugs, including opiates (Kirby et al., 1999; Kirby and Petry,

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2004), cocaine (Coffey et al., 2003; Heil et al., 2006), alcohol (Petry, 2001a) tobacco (Baker et al., 2003; Bickel et al., 1999; Odum et al., 2002; Reynolds et al., 2004), and also for poly-drug use (Businelle et al., 2010; Petry and Casarella, 1999). Several studies have observed even greater discounting among individuals with SUDs that present other comorbidities, such as gambling among alcohol-dependent individuals (Petry, 2001b; Petry and Casarella, 1999; Stea et al., 2011), obesity among adolescent cigarette smokers (Fields et al., 2011), or risky behaviors for HIV such as needle sharing among opiate-dependent individuals (Odum et al., 2000). Additionally, emerging evidence suggests that delay discounting may serve as an important indicator of both treatment success and drug use onset. For example, baseline discounting predicted relapse in smokers (Dallery and Raiff, 2007; Yoon et al., 2007) and treatment outcomes in cocaine-dependent individuals (Washio et al., 2011) and in adolescents receiving treatment for marijuana abuse or dependence (Stanger et al., 2012). Likewise, baseline delay discounting predicted which individuals would initiate cigarette smoking in a longitudinal study tracking high-school students (Audrain-McGovern et al., 2009).

Nevertheless, less is known about delay discounting rates among subjects with different SUDs Businelle et al. (2010) observed that heavy smokers' delay discounting was similar to that of other substance-dependent individuals. In contrast, Kirby and Petry (2004) observed greater discounting among cocaine- and heroin-dependent individuals compared to alcohol-dependent individuals. Moreover, although illegal poly-drug use is common among individuals with SUDs (Grant et al., 2004; Smith et al., 2011), these participants tend to be excluded from studies (Kirby and Petry, 2004). Only Businelle et al. (2010) reported that people dependent upon multiple drugs did not discount delayed rewards more than those dependent on only one substance. However, the value of these results is limited by the heterogeneity of the poly-drug users group in terms of type and number of SUDs.

Although tobacco use (Baker et al., 2003; Bickel et al., 1999; Mitchell, 1999; Ohmura et al., 2005) has been associated with higher rates of discounting, illicit drug users who also smoke cigarettes are rarely analyzed as multiple drug users in delay discounting research. Taking into account that approximately 70% of substance-dependent individuals smoke (Richter et al., 2002), and that rates of smoking are 3- to 4-fold greater among cocaine-dependent individuals than in the general population (Budney et al., 1993; Gorelick et al., 1997; Kalman et al., 2005; Lasser et al., 2000; Patkar et al., 2006, 2002), most studies have ignored the potential effects of smoking on delay discounting. When the drug of choice is not tobacco, smoking has either been controlled (Kirby and Petry, 2004) or ignored as a potential confounding variable (Heil et al., 2006; Kirby and Petry, 2004; Petry, 2001a). As a result, the relation between different combinations of multiple SUDs including tobacco and delay discounting remains unclear. More accurate knowledge of whether dependence on one or on several substances are related to differential performance on the delay discounting task is needed for a better understanding of this phenomenon.

The present study sets out to address some of the limitations of previous studies, with the goal of comparing performance on a delay discounting task across four groups: cocaine- and nicotine-dependent participants, cocaine-dependent only participants, nicotine-dependent only participants, and non-dependent controls.

2. Materials and methods

2.1. Participants

Participants consisted of a subset of individuals originally recruited for two studies related to the treatment of cocaine

($N=47$) and nicotine dependence ($N=30$) as well as non-dependent controls recruited throughout the community ($N=30$). All the participants were males and residents of Spain.

Inclusion criteria for all participants were being under 18 years old, reporting any psychiatric disorder that would prevent their carrying out the delay discounting task, and meeting *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; DSM-IV) criteria for drug abuse or dependence other than the ones described below.

Inclusion criteria for cocaine- and nicotine-dependent participants were as follows: meeting DSM-IV criteria for cocaine and nicotine dependence, smoking 10 or more cigarettes per day, and having used cocaine in the last 3 months. Inclusion criteria for cocaine-dependent only participants were meeting DSM-IV criteria for cocaine dependence and having used cocaine in the last three months. Inclusion criteria for nicotine-dependent only participants were meeting DSM-IV criteria for nicotine dependence and smoking a minimum of 10 cigarettes per day. Inclusion criteria for non-dependent control participants were not meeting DSM-IV criteria for any drug abuse or dependence. As the cocaine-dependent participants recruited were all males, the nicotine-dependent only group and the control group were matched in gender.

This study was approved by the Institutional Review Board of the University of Oviedo, and informed consent was obtained from all participants prior to study initiation.

2.2. Procedure and instruments

The delay discounting task was presented to participants via a laptop computer running the Windows operating system. Overall, the task took approximately 10 min to complete for each participant. Participants were instructed how to interact with the delay discounting program and informed that they would not receive any of the monetary amounts presented, but they were to respond as if the choices were real. Participants were presented with a choice between €1000 after a fixed delay versus various amounts of money available immediately using an adjusting-amounts procedure (Holt et al., 2012). The delays values were one day, one week, one month, six months, one year, five years and 25 years. The delays were presented in an ascending order for all the participants. The value of the immediate monetary option ranged from €5 to €1000 in €5 increments and was adjusted via a titrating procedure that honed in on the indifference point based on the participants' responses. The titration procedure took the lower and upper limit of possible values (initial €0 and €1000) and divided this total range randomly by 2, 3, or 4 to obtain an interval value. The value of the immediate option was one interval value above or below the upper and lower limits. If the immediate value was outside €0 and €1000, another value was randomly chosen. New lower and upper limits were chosen based on the participant's response, adjusting the total range, and the titration process was repeated. Note that based on the possible values presented, the total range could occasionally increase if they chose an option outside of the total range. Once the total range was at or less than €40, the average of the upper and lower limits was taken as the indifference point, and the next delay was presented.

DSM-IV diagnosis were assessed with the Spanish version of the Structure Clinical Interview for DSM (SCID) (First et al., 1999).

2.3. Data analysis

In order to assess k values for each individual, the hyperbolic model was fitted to each subject's delay discounting data (i.e., indifference points) with nonlinear regression (SAS, PROC NLIN). Goodness of fit was evaluated on the basis of model R^2 s. As the distribution of k values was skewed, frequency distributions for each group were generated from log k values.

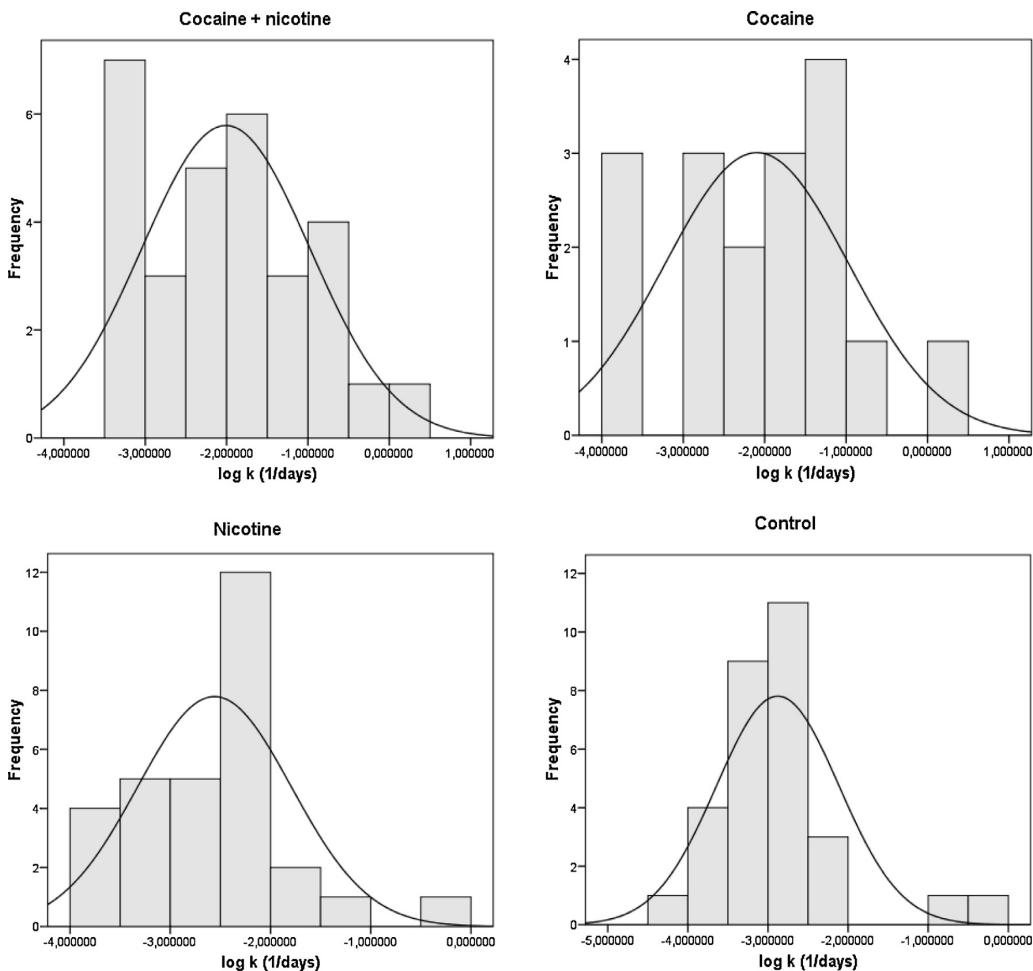


Fig. 1. Frequency distribution of $\log k$ across groups. Top panel shows the frequency distribution of the $\log k$ values for the cocaine and nicotine-dependent group and for the cocaine-dependent only group. Bottom panel shows the frequency distribution of the $\log k$ values for the nicotine-dependent group and for the control group. Units for $\log k$ variable are days^{-1} .

The k values for each group were assessed by fitting the hyperbolic model to all the indifference points for a given group using nonlinear regression via GraphPad Prism 5.0 (Graphpad Software, San Diego, CA). Comparisons of k values between specific groups were completed using global non-linear regression, which assessed whether the obtained indifference points from two groups are better characterized by one k value (i.e., delay discounting in each group is similar) or two k values (i.e., delay discounting is different between groups) (Motulsky and Christopoulos, 2004). The null hypothesis is that the two groups share the same k value, and the alternative hypothesis is that the k values are different. Using the extra sum-of-squares F test, Prism was used to assess the goodness of fit of two-models (shared vs. distinct k values) as assessed by sum-of-squares and adjusting for differences in the number of degrees of freedom. Similar analyses were conducted to compare delay discounting for a subset of participants in an attempt to control for differences in education. Although often not presented, the units of k are $1/\text{days}$. For ease of comparisons with previous results in the literature and to potentially make delay discounting results more accessible, we also present the ED50 value for the primary comparison. ED50 is the delay that is effective in discounting the subjective value of the delayed reinforcer (€1000) by 50% (Yoon and Higgins, 2008).

Comparison of sociodemographic and substance-use characteristics between groups was performed using Student's t tests (after Levene's correction for inequality of variance) and one-way between groups ANOVAs using SPSS (V15; SPSS, Inc., Chicago, IL). Significance for all statistical comparisons was defined at $p \leq .05$.

3. Results

Table 1 shows comparison of demographic and substance use-related characteristics among groups. No statistically significant differences were found between groups in any of the variables analyzed with the exception of years of education. Specifically, the cocaine- and nicotine-dependent group and the cocaine-dependent only group reported fewer years of education compared to both the nicotine-dependent only group and the control group.

Eq. (1) provided an appropriate fit to individual participant's delay discounting data. The median R^2 was .93, and 73.3% of subjects had an R^2 greater than .80. Fig. 1 shows the frequency distribution of $\log k$ values for each group.

Comparisons of delay discounting across groups are depicted in Fig. 2. Curves (top panel) and bars (bottom panel) are based on best fit k values to group indifference points. Plots of median indifference points for each group as a function of

Table 1

Comparison of demographic and substance use-related characteristics among groups.

Characteristic	Group				Statistic value	p
	Cocaine + nicotine	Cocaine	Nicotine	Control		
Demographic characteristics ^a						
Age	32.5 (0.8)	33.6 (1.3)	28.2 (1.7)	28.5 (2.1)	2.49 ¹	.07
Education (years)	10.9 (0.5) ^a	10.7 (0.7) ^a	14 (0.4) ^b	14 (0.5) ^b	12.73 ¹	<.001
Monthly income, Euros	718 (105)	940 (281)	800 (115)	863 (124)	0.38 ¹	.76
Tobacco use-related characteristics ^a						
Cigarettes per day	14.6 (1.8)	—	15.3 (1.1)	—	0.31 ²	.75
Cocaine use-related characteristics						
Intranasal administration (%)	96.7	100	—	—	0.58 ³	.44
Age at onset of regular cocaine use ^a	19 (0.6)	18 (0.5)	—	—	1.19 ²	.24
Duration of regular cocaine use, years ^a	10 (0.9)	10.3 (1.2)	—	—	-0.21 ²	.83
Time since last use, days ^a	33.1 (5.9)	45.1 (8.6)	—	—	-1.181 ²	.24

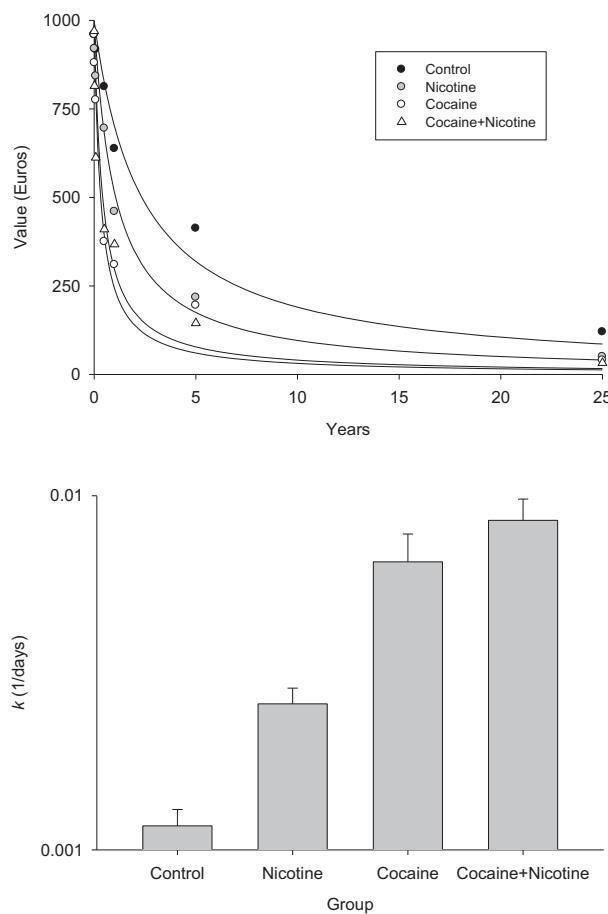
Means in the same row with different subscripts differ significantly at $p < .05$ in the Tukey honestly significant difference comparison; Statistic used: ¹ANOVA; ²Student's *t*;³Chi-squared.Bold: statistically significant at $p < .05$ ^a Mean (\pm standard error).

Fig. 2. Comparisons of delay discounting across groups. Top panel: Curves represent best fitting hyperbolic functions based on k values to group indifference points for each group. The symbols represent the median indifference points for each delay in each group: white triangles for the cocaine- and nicotine-dependent group, white circles for the cocaine-dependent only group, gray circles for the nicotine-dependent only group and black circles for the control group. Bottom panel: Bars represent best fit group k values and standard errors. The Y axis is on a log scale.

delay intervals are also included. The curves represent best fitting hyperbolic functions based on k values derived from global non-linear regression for each group. The symbols indicate the median indifference points for a given delay in the cocaine and nicotine-dependent group (white triangle), the cocaine-dependent only group (white circle), the nicotine-dependent only group (gray circle) and the control group (black circle). Overall, greater discounting was observed in the cocaine- and nicotine-dependent group ($k = 0.0085 \text{ days}^{-1}$, $SE = 0.0012 \text{ days}^{-1}$) and the cocaine-dependent only group ($k = 0.0065 \text{ days}^{-1}$, $SE = 0.0012 \text{ days}^{-1}$), followed by the nicotine-dependent only group ($k = 0.0026 \text{ days}^{-1}$, $SE = 0.0003 \text{ days}^{-1}$), and then the control group ($k = 0.0012 \text{ days}^{-1}$, $SE = 0.0001 \text{ days}^{-1}$). Note that steeper curves and higher bars indicate greater discounting. Alternatively, it took 0.3 years in the cocaine- and nicotine-dependent groups to discount the delayed options (i.e., €1000) by 50%. In comparison, the cocaine-dependent only group took 0.4 years, the nicotine-dependent only group took 1.1 years, and the control group took 2.3 years.

Global non-linear regression showed statistically significant differences in group delay discounting between all group comparisons with the exception of the cocaine- and nicotine-dependent group and the cocaine-dependent only group comparison ($F(1, 327) = 0.72, p = .394$). Delay discounting was significantly greater in the cocaine- and nicotine-dependent group than in the nicotine-dependent only group ($F(1, 418) = 31.34, p < .001$) and also than in the control group ($F(1, 418) = 83.95, p < .001$). Similarly, delay discounting in the cocaine-dependent only group was significantly greater than in the nicotine-dependent only group ($F(1, 327) = 14.53, p < .001$) and also than in the control group ($F(1, 327) = 48.61, p < .001$). Lastly, delay discounting in the nicotine-dependent only group was significantly greater than that of the control group ($F(1, 418) = 20.16, p < .001$).

A secondary analysis was conducted on a subset of participants in an attempt to control for observed differences in education levels. This subset ($N = 79$) excluded the seven participants with the highest educational achievement in the nicotine-dependent group and in the control group, and the seven participants with lowest education levels in the cocaine- and nicotine-dependent group and the cocaine-dependent group. By excluding the top and lowest participants, education level was no longer significantly different across the four groups. Observed effects (data not shown) mirrored those observed in the main comparisons with original complete groups. Additionally, k values were compared between each full group and their subset (original complete group vs. subset controlling for education) and no significant differences in delay discounting were observed (data not shown).

4. Discussion

The goal of this study was to compare delay discounting across groups of individuals dependent on cocaine and nicotine, on cocaine only, on nicotine only, and non-dependent controls. There are three key findings in this study. First, delay discounting was greater in all the substance-dependent groups compared to the non-dependent control group. Second, both the cocaine-dependent and cocaine- and nicotine-dependent groups exhibited greater discounting than the nicotine-dependent group. Third, delay discounting for the cocaine- and nicotine-dependent group did not differ from the cocaine-dependent group.

In line with those of previous studies (Coffey et al., 2003; Heil et al., 2006; Kirby and Petry, 2004), our results indicate that delayed reinforcers lose value more rapidly in illegal substance-dependent individuals (cocaine plus nicotine and cocaine only in the present study) than in non-dependent controls. Also higher rates of discounting among nicotine-dependent smokers by comparison with matched controls have consistently been found in previous research (Baker et al., 2003; Bickel et al., 1999; Reynolds et al., 2004; Mitchell, 1999). Although delay discounting is often characterized as a relatively stable personality trait (Odum, 2011), greater impulsivity may also be understood as both a predisposing risk factor for addictive behavior and a consequence of the addictive disorders (MacKillop et al., 2011). Future research may provide information of the role of the impulsive behavior throughout the entire addictive process, from early drug use to relapse or cessation, considering delay discounting as a specific target for therapeutic attempts (Odum, 2011).

The second finding of the present study points to a differential effect on performance in the delay discounting task as a function of the type of substance of dependence. In our study, individuals dependent on cocaine (from both cocaine and nicotine, and cocaine only groups) discount delayed rewards significantly more than those dependent on nicotine only. Businelle et al. (2010) reported that heavy smokers and other substance-dependent individuals did not differ in discounting rates and Petry and Casarella (1999) asserted that type of drug use is probably not related to higher discounting rates. These studies have the main limitation of including different SUDs (cocaine, heroin or alcohol) in the same group when comparing them with smokers or pathological gamblers, making it impossible to determine whether delay discounting rates are actually similar or not across subjects with different SUDs. In contrast, when discounting between those with different SUDs were separated into independent groups, differences as a function of type of drug dependent were found (Johnson et al., 2010; Kirby and Petry, 2004). These current results suggest that delay discounting rates may not be the same for all subjects with SUDs. This would be in line with a continuous conception of delay discounting, rather than the notion of a more categorical difference between non-dependent and drug-dependent individuals. Delay discounting reflects universal behavioral process build on subject learning history that may both contribute and be affected for other behaviors such as drug use.

The third finding of this study revealed that there is no cumulative effect on the delay discounting task performance depending on the number of SUDs. The subjects in the cocaine-dependent only group did not differ from those dependent on both cocaine and nicotine. In line with the previous result, the type of SUD appears to be more relevant than the number of SUD. This finding is consistent with Businelle et al. (2010) study, reporting that being dependent on multiple substances did not modify rates of discount.

This study has some limitations that should be mentioned. First, the sample is made up exclusively of men, and this may reduce the potential for generalization of the results. Delay discounting studies among non-dependent individuals have observed significant

differences in delay discounting between males and females (Kirby and Marakovic, 1996). However, most studies with substance abusers have not reported significant gender differences in delay discounting (Businelle et al., 2010; Epstein et al., 2003; Reynolds et al., 2004; Kirby and Petry, 2004). Second, the small sample size in the cocaine-dependent group may have restricted the detection of significant differences among this group and the cocaine and nicotine-dependent group. Third, despite all cocaine-dependent participants met DSM-IV criteria for this diagnosis, time since last use was variable across the sample (from 0 to 90 days) and due to sample size, this variable was not controlled. Fourth, although we matched or controlled the differences among groups in age, income and years of education, there are other variables not recorded for this study that may have affected the results obtained, such as family history of substance dependence (Petry et al., 2002) or history of depression (Yoon et al., 2007). Similarly, previous studies have found that differences in intelligence (Olson et al., 2007; Shamosh et al., 2008) and education achievement (Jaroni et al., 2004; Silva and Gross, 2004) could be related with delay discounting rates. In the current study, sensitivity analyses for a subset of participants equally educated were conducted and same results as those with the entire sample were observed but no intelligence measure was collected.

5. Conclusion

Overall, and despite these limitations, these findings suggest that delay discounting rates differs depending on the type of SUD but not on the number of SUDs. However, there is a need for further research comparing delay discounting rates for a wider range of substances than in the present study.

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Conflict of interest

All the authors declare that they have no conflicts of interest.

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3.2. Artículo 2

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Resumen: Current cigarette smokers exhibit greater delay discounting relative to ex-smokers. However, few studies have assessed longitudinal changes in delay discounting and cigarette smoking. The purpose of this study was to assess changes in delay discounting of hypothetical monetary rewards and smoking among treatment-seeking smokers ($N = 80$) at baseline, after 6 weeks of behavioral treatment, and at 12-month follow-up. Results showed no changes in delay discounting in either smokers or abstainers at the end-of-treatment. In contrast, at 12-month follow-up, significant decreases in delay discounting were observed in abstainers while delay discounting remained the same for smokers. To our knowledge, this is the first study to observe significant decreases in delay discounting following prolonged smoking abstinence. Such findings provide evidence that delay discounting may have more state-like characteristics than previously believed

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Decreased delay discounting in former cigarette smokers at one year after treatment



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HIGHLIGHTS

- We examine changes in delay discounting rates as a function of smoking status.
- Delay discounting does not change as a function of initial abstinence.
- Delay discounting significantly decreased among abstainers by 12-months follow-up.
- Cigarette smoking could cause reversible changes in discounting rates.

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ABSTRACT

Current cigarette smokers exhibit greater delay discounting relative to ex-smokers. However, few studies have assessed longitudinal changes in delay discounting and cigarette smoking. The purpose of this study was to assess changes in delay discounting of hypothetical monetary rewards and smoking among treatment-seeking smokers ($N = 80$) at baseline, after 6 weeks of behavioral treatment, and at 12-month follow-up. Results showed no changes in delay discounting in either smokers or abstainers at the end-of-treatment. In contrast, at 12-month follow-up, significant decreases in delay discounting were observed in abstainers while delay discounting remained the same for smokers. To our knowledge, this is the first study to observe significant decreases in delay discounting following prolonged smoking abstinence. Such findings provide evidence that delay discounting may have more state-like characteristics than previously believed.

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1. Introduction

Delay discounting characterizes how a reinforcer loses value as the delay to its receipt increases (Bickel & Marsch, 2001; Reynolds, 2006). Delay discounting provides an explanation as to why some individuals may choose a smaller, more immediate reinforcer (e.g., smoking a cigarette and consuming a drug) over a relatively larger, delayed one (e.g., improved health, financial stability, and social standing). In this manner, delay discounting provides an operational definition of impulsivity that may underscore certain aspects of drug use. In human studies, delay discounting is typically assessed using an adjusting-delay procedure in which an individual is presented with multiple choices (usually, hypothetical monetary rewards) between a relatively smaller, more immediate reinforce vs. a relatively larger, more delayed one (Rachlin, Rainieri, & Cross, 1991). The magnitude of the smaller, immediate choice is adjusted until the two choices are deemed subjectively equal (i.e. an

indifference point is reached), and the process is repeated at different delays (Rachlin et al., 1991).

Greater delay discounting (i.e., greater impulsivity) has been reliably observed among those with substance use disorders (SUDs) compared to matched controls across a variety of common drugs of abuse (Baker, Johnson, & Bickel, 2003; Coffey, Gudleski, Saladin, & Brady, 2003; García-Rodríguez, Secades-Villa, Weidberg, & Yoon, 2013; Heil, Johnson, Higgins, & Bickel, 2006; Kirby & Petry, 2004; Kirby, Petry, & Bickel, 1999; Petry, 2001; Reynolds, Richards, Horn, & Karraker, 2004). Indeed, delay discounting appears to be fundamentally tied with SUDs, with greater discounting associated with drug use initiation, maintenance, severity, and relapse (Madden & Johnson, 2010). Overall, such evidence suggests that delay discounting should be considered as a candidate behavioral marker for addiction (Bickel, Koffarnus, Moody, & Wilson, 2014).

Among those with SUDs, cigarette smokers have received the most attention in regard to delay-discounting research (Yi, Mitchell, & Bickel, 2010). Previous research has demonstrated that smokers exhibit greater discounting relative to matched, non-smoking controls (Baker et al., 2003; Bickel, Odum, & Madden, 1999; Mitchell, 1999; Wing, Moss, Rabin, & George, 2012). Greater discounting is associated with

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earlier age of smoking initiation and earlier adoption of regular smoking (Audrain-McGovern et al., 2009; Kollins, 2003). Additionally, greater delay discounting is associated with smoking severity in terms of the amount of nicotine intake per day (Ohmura, Takahashi, & Kitamura, 2005), and with increased risk of smoking relapse in both laboratory studies (Dallery & Raiff, 2007; Mueller et al., 2009) and clinical trials (MacKillop & Kahler, 2009; Sheffer et al., 2012; Yoon et al., 2007).

Whether delay discounting is a stable trait or a changing state variable is currently the focus of extensive research (Audrain-McGovern et al., 2009; de Wit, 2009). In a seminal series of studies comparing current-, former-, and never-smokers, greater discounting was observed in the current smokers, whereas delay discounting among former- and never-smokers was basically indistinguishable (Baker et al., 2003; Bickel et al., 1999; Odum, Madden, & Bickel, 2002). Such results begged the question whether discounting among former smokers decreased as a result of smoking abstinence, if their smoking abstinence was the result of preexisting lower discounting rates, or a third variable (Black & Rosen, 2011; de Wit, 2009; Yi et al., 2008). However, the cross-sectional nature of these studies does not permit a way to determine the causal relationship between smoking status and delay discounting (Audrain-McGovern et al., 2009).

Delay discounting has presented state-like characteristics following acute smoking deprivation (Field, Santarcangelo, Sunnall, Goudie, & Cole, 2006; Mitchell, 2004; Yi & Landes, 2012) and acute opioid withdrawal (Giordano et al., 2002). Similarly, another study observed that significant decreases in discounting among smokers follow five days of contingency management treatment (Yi et al., 2008). In contrast, relatively longer longitudinal studies have observed delay discounting to exhibit trait-like characteristics despite changes in smoking status. For example, in a longitudinal cohort study tracking adolescents into young adulthood (15 to 21 years old), those who eventually became smokers exhibited greater baseline delay discounting, but no significant changes in discounting were observed over time (Audrain-McGovern et al., 2009). Similarly, two studies found no changes in delay discounting among smokers despite prolonged periods of smoking abstinence (Yoon, Higgins, Bradstreet, Badger, & Thomas, 2009; Yoon et al., 2007). Taken together, the results from longitudinal studies support a trait-like interpretation of delay discounting regarding longer-term changes in smoking status. Similar findings have been observed among different populations of individuals with SUDs after receiving formal treatment with longer follow-ups (Landes, Christensen, & Bickel, 2012; Peters, Petry, LaPaglia, Reynolds, & Carroll, 2013).

To our knowledge, no studies have directly assessed longitudinal changes in delay discounting associated with quitting smoking among treatment-seeking smokers. Moreover, most of the previous studies conducted with cigarette smokers have relied on follow-up assessments shorter than 1 month, which may prevent the detection of changes in delay discounting rates following longer periods of abstinence. We sought to build on prior research and conduct a longitudinal assessment of changes in delay discounting and smoking status among treatment-seeking smokers in a community setting. Therefore, the primary goal of the current study was to analyze if delay discounting rates change as a function of smoking status at the end-of-treatment and at 12-month follow-up.

2. Materials and methods

2.1. Participants

A total of 96 subjects were enrolled in the clinical trial. As the purpose of the current study was to assess changes in delay discounting as a function of smoking status, only participants who completed treatment and the 12-month follow-up were included in the final analysis ($N = 80$). Of the 16 subjects excluded from the final analysis, 5 dropped out of treatment and 11 missed the 12-month follow-up assessment. No significant differences were observed between excluded subjects

and those who were included in the final analysis neither in their sociodemographic and smoking-related characteristics nor in their baseline delay discounting.

Inclusion criteria consisted of participants being at least 18 years old and self-reporting smoking at least 10 or more cigarettes per day for at least one year. Participants were excluded if they presented a psychiatric disorder (including substance use disorder besides nicotine dependence) or were receiving any other smoking cessation treatment. This study was approved by the Institutional Review Board of the University of Oviedo, and informed consent was obtained from all participants prior to study initiation.

The final sample for the present study consisted of 80 adult smokers (66.3% women). Mean age was 38.90 ($SD = 13.12$) years and mean years of education were 13.19 ($SD = 2.42$) years. The percentage of participants who were married was 46.3. Participants smoked a mean of 19.33 ($SD = 8.69$) cigarettes per day during an average of 19.99 ($SD = 11.49$) years. They reported a mean of 2.28 ($SD = 2.27$) previous quitting attempts. Mean breath carbon monoxide (CO) was 13.59 ($SD = 6.51$) ppm, and the mean score on the Fagerström Test for Nicotine Dependence (FTND) was 5.11 ($SD = 2.20$), indicating medium nicotine dependence (Fagerstrom, Heatherton, & Kozlowski, 1990).

2.2. Treatment

Participants received group-based, cognitive-behavioral treatment (CBT) described in previous studies (Secades-Villa, Alonso-Perez, García-Rodríguez, & Fernández-Hermida, 2009; Vázquez & Becoña, 1999). To summarize, CBT groups consisted of five to six participants. Session duration was approximately 1 h and sessions were carried out once per week for six weeks. CBT sessions were highly structured and included: information about tobacco, behavioral contract, self-monitoring and graphical representation of cigarette smoking, nicotine fading, stimulus control, strategies for controlling nicotine withdrawal symptoms, physiological feedback consumption, training in alternative behaviors, social reinforcement of objective completion and abstinence, and relapse prevention strategies.

2.3. Assessments

Participants' baseline socio-demographic and smoking-related characteristics were assessed during the intake session, which took approximately 1 and a half hour. The FTND (Heatherton, Kozlowski, Frecker, & Fagerstrom, 1991) was used to assess nicotine dependence in addition to the Structured Clinical Interview (SCID-I) of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR) (American Psychiatric Association, 2000).

The smoking abstinence criteria were defined as presenting a breath CO level ≤ 4 ppm (Lamb, Kirby, Morral, Galicka, & Iguchi, 2010; Meredith, Grabinski, & Dallery, 2011; Perkins, Karelitz, & Jao, 2013; Romanowich & Lamb, 2010) and providing a self-report of smoking abstinence (not even a puff) for the past 24 h at post-treatment (6 weeks) and the last 7 days at 12-month follow-up (Cavallo et al., 2007; Hughes et al., 2003). Breath CO was assessed using a Micro⁺ Smokerlyzer (Bedfont Scientific Ltd., Harrietsham, Kent).

Delay discounting was assessed at intake, at the end-of-treatment (6 weeks), and at 12-month follow-up. The delay discounting task was presented to the participants via a laptop running the Windows Operating system. Overall, the task took approximately 10 min to complete for each participant. Participants were instructed how to interact with the delay discounting program and informed that they would not receive any of the monetary amounts presented, but they were to respond as if the choices were real. Previous studies have demonstrated that participants respond similarly during delay discounting tasks for both real and hypothetical monetary values (Johnson & Bickel, 2002; Lawyer, Schoepflin, Green, & Jenks, 2011). Details regarding the delay discounting task have been published elsewhere (García-Rodríguez

et al., 2013). Participants were presented with a choice between €1000 after a fixed delay versus various amounts of money available immediately using an adjusting-amount procedure (Holt, Green, & Myerson, 2012). The delay values were one day, one week, one month, six months, one year, five years and 25 years. The delays were presented in an ascending order for all the participants. The value of the immediate monetary option ranged from €5 to €1000 in €5 increments and was adjusted via a titrating procedure that honed in on the indifference point based on the participants' responses.

2.4. Data analysis

Indifference points were fitted to the Mazur's (1987) hyperbolic equation (Green, Fry, & Myerson, 1994; Kirby, 1997; Myerson & Green, 1995):

$$V = A/(1 + kD). \quad (1)$$

The equation describes how the subjective value (V) of a reinforcer of some amount (A) is discounted as a function of delay (D) to receiving it (Mazur, 1987). The free parameter k describes the rate of discounting, with higher values of k indicating greater discounting and impulsivity (Reynolds, 2006).

In order to assess k values for each individual, the hyperbolic model was fitted to each subject's delay discounting data (i.e., indifference points) with nonlinear regression (SAS, PROC NLIN). Goodness of fit was evaluated on the basis of model R^2 's. Because the distribution of estimated k values was skewed, analyses were performed on log-transformed k values.

A mixed between-within subjects ANOVA was conducted to assess the impact of smoking status (smokers vs. abstainers) on participants' delay discounting rates across two time periods (intake and end-of-treatment). The same analysis was performed across the intake and 12-month follow-up.

Effect sizes of principal comparisons were calculated using partial eta squared ($\eta^2 p$). The statistics software used was SPSS (V15; SPSS, Inc., Chicago, IL). Significance for all statistical comparisons was defined at $p \leq .05$.

3. Results

Eq. (1) described above provided an appropriate fit to subjects' delay discounting data. The median R^2 value was .90, and 72.1% of subjects had an R^2 value greater than .80.

3.1. Delay discounting rates at the end-of-treatment

Sixty-one percent of the participants were abstinent at the end-of-treatment. There was no significant effect of time from intake to the end-of-treatment in the whole sample (Wilks' Lambda = .985, $F(1, 78) = 1.204, p = .276, \eta^2 p = .015$). Similarly, there was no significant effect of smoking status ($M_{smokers} = -2.7033, SD = 0.7284; M_{abstainers} = -2.9452, SD = 0.8200$) at the end-of-treatment ($F(1, 78) = 1.653, p = .202$). Fig. 1 shows the delay discounting curves in the intake and the end-of-treatment for both smokers (upper panel) and abstainers (lower panel). Curves represent the best fitting hyperbolic functions based on k values derived from Eq. (1). Symbols represent the median indifference points for a given delay at each assessment. Note that steeper curves indicate greater discounting.

3.2. Delay discounting rates at 12-month follow-up

Thirty-five percent of the participants were abstinent at 12-month follow-up. There was no significant effect of time from intake to 12-month follow-up in the whole sample (Wilks' Lambda = .980, $F(1, 78) = 1.594, p = .211$). However, there was a significant main effect of smoking status at 12-month follow-up ($F(1, 78) = 5.601,$

$p = .020, \eta^2 p = .067$). Specifically, participants who were smoking at 12-month follow-up exhibited greater delay discounting ($M = -2.6370, SD = 0.8704$) compared to abstainers ($M = -3.1597, SD = 0.7720$). Fig. 2 compares the delay discounting curves at intake and 12-month follow-up for both smokers (upper panel) and abstainers (lower panel).

4. Discussion

To our knowledge, this is the first study to examine longitudinal changes in delay discounting and smoking status among treatment-seeking smokers. The main and novel finding of the present study is that delay discounting significantly decreased among abstainers at 12-month follow-up, while no changes in delay discounting were observed among smokers throughout the study.

Our results support previous evidence that delay discounting does not change right after individuals quit smoking, as a function of initial abstinence (Dallery & Raiff, 2007; Yoon et al., 2009), and suggest that a longer period of abstinence may be necessary before changes in discounting rates can be observed.

Although no significant changes in delay discounting were observed at the end-of-treatment between smokers and abstainers, delay discounting was significantly decreased among abstainers by 12-month follow-up, while no significant changes were observed among those that continued to smoke. These results stand out from previous findings supporting the characterization of delay discounting as a trait-like characteristic that remains relatively stable despite changes in smoking status (Audrain-McGovern et al., 2009; Yoon et al., 2007) and suggests that delay discounting may have more state-like characteristics (Field et al., 2006; Giordano et al., 2002; Reynolds, 2004; Yi et al., 2008). Therefore, our results are in line with previous considerations regarding cigarette smoking as a behavior which could cause a reversible change in discounting rates (Bickel et al., 1999) and support the hypothesis that decreases in delay discounting are due at least in part to the extended smoking abstinence and not to previously existing differences in delay discounting (Audrain-McGovern et al., 2009; Yoon et al., 2007, 2009). Several mechanisms may contribute to explaining decreased delay discounting among participants who quit smoking at longer-term follow-ups. First, engagement in healthy behaviors has been inversely associated with delay discounting rates (Bradford, 2010; Melanko & Larkin, 2013). For example, abstinence from smoking can promote changes similar to adopting healthier lifestyles (e.g., having adequate dietary habits and body mass index (BMI) or practicing regular exercise) and these changes may result in a decreased delay discounting (Jang et al., 2012; Nagaya, Yoshida, Takahashi, & Kawai, 2007). Second, according to the competing neurobehavioral decision system hypothesis of addiction (Bechara, 2005; Bickel et al., 2007), abstinence from smoking may promote a reduction in the activation of the impulsive decision system, and a parallel increase in the activation of the executive system, which could lead to a reduction in delay discounting rates. SUDs result from a hyperactive impulsive decision system (e.g., limbic and paralimbic regions) which promotes choices of immediate reinforcement and a hypoactive executive system (e.g., prefrontal cortex) that undermines planning and choices of delayed reinforcement (Bickel, Yi, Kowal, & Gatchalian, 2008). Finally, smokers show processing bias towards smoking-related cues when compared to former smokers (Littel, Franken, & Van Strien, 2009; Munafó, Mogg, Roberts, Bradley, & Murphy, 2003), and delay discounting rates and measures of processing bias have been positively correlated (Field, Christiansen, Cole, & Goudie, 2007; Murphy & Garavan, 2011). It is possible that prolonged abstinence results in some degree of reversal of processing bias towards smoking cues due to the creation of new, null associations for smoking-related cues (Munafó et al., 2003). Hence, this reversal may result in a decrease in delay discounting rates among smokers who are long-term abstinent. These mechanisms by which discounting rates are susceptible of being modified are

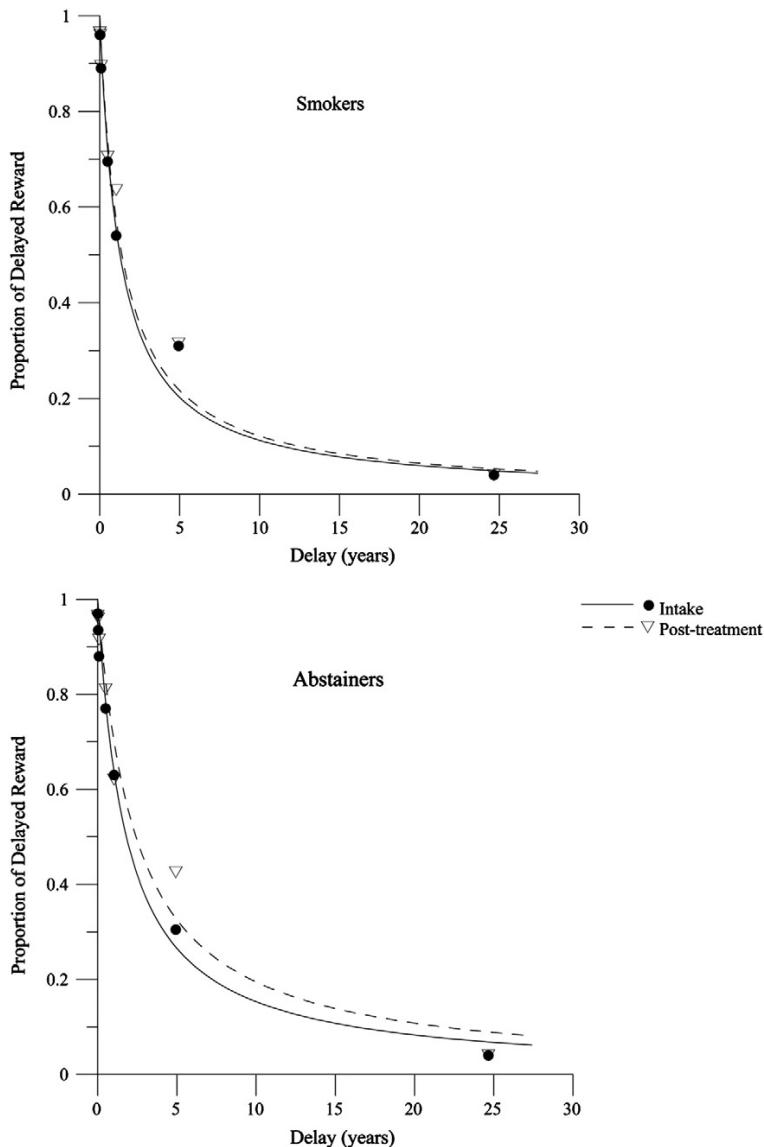


Fig. 1. Delay discounting curves in the intake and post-treatment.

not mutually exclusive and may take time to occur. This could explain why decreases in delay discounting were detected only at 12-month follow-up and not at the end-of-treatment. However, the present study does not address if these potential mechanisms described above contribute to the decreases in delay discounting observed in the current study. Future studies should address whether and how such mechanisms result in changes in delay discounting after prolonged smoking abstinence.

Some clinical implications of this study should be considered. If current smokers discount delayed consequences more steeply than abstinent smokers (Bickel et al., 1999; Odum et al., 2002), treatments for smoking cessation must focus on the short-term consequences of smoking or on arranging short-term mediating consequences rather than on the long-term consequences of smoking (Odum et al., 2002). In this regard, contingency management (CM) has proven to be an efficacious treatment for promoting abstinence among both non-treatment-seeking (Alessi, Badger, & Higgins, 2004; Heil, Tidey, Holmes, Badger, &

Higgins, 2003; Roll & Higgins, 2000) and treatment-seeking smokers (Dallery, Glenn, & Raiff, 2007; Lamb, Morral, Kirby, Iguchi, & Galicka, 2004). However, many participants often relapse once CM is discontinued. Additional research is needed to explore novel ways in which CM-induced abstinence continues once CM treatment is no longer provided (Ledgerwood, 2008). Also, there is recent evidence that CM treatment results in a decrease of delay discounting among opioid-dependent individuals (Landes et al., 2012) so future studies should assess the extent to which CM effectiveness produces analogous decreases in delay discounting among smokers.

The current study has several potential limitations that may affect the interpretation of the results and point to future research lines. First, we had no control group of matched never smokers. This group would allow us to assess if delay discounting rates of smokers who were abstinent at 12-month follow-up actually decreased to pre-smoking levels. Second, the current study defined abstinence using a combination of biological measures and gold-standard criteria for 12-

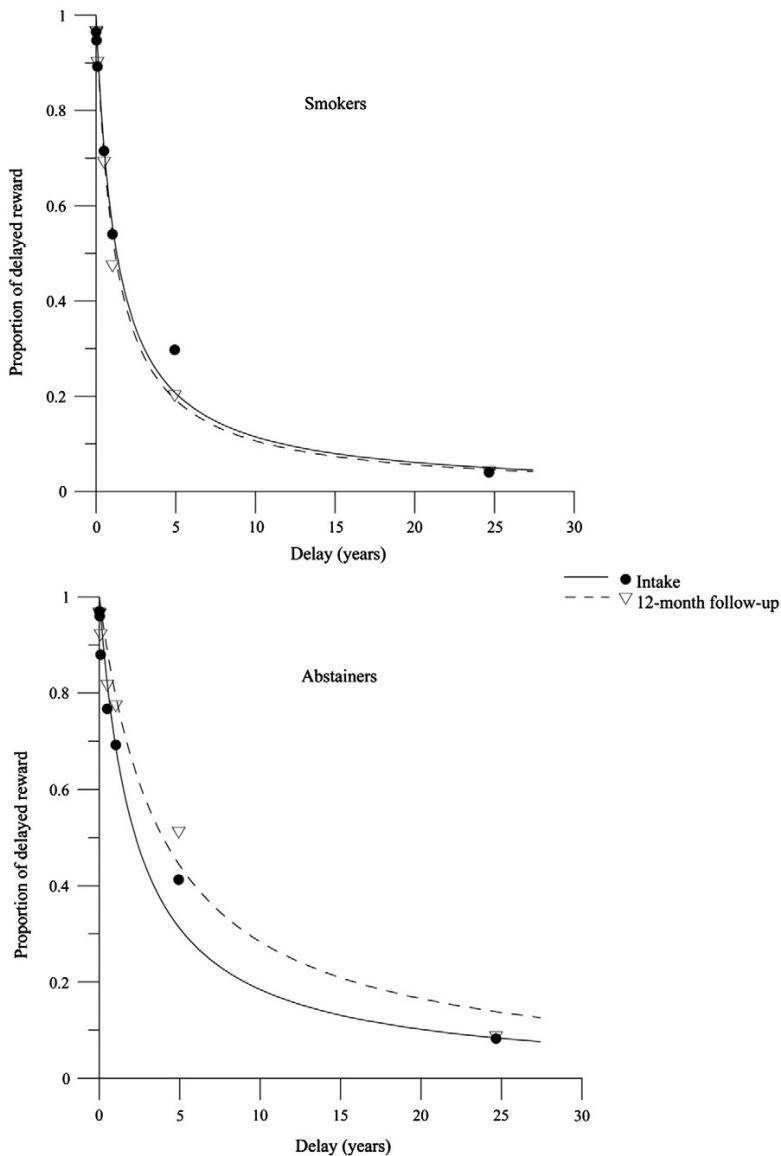


Fig. 2. Delay discounting curves in the intake and 12-month follow-up.

month follow-up abstinence, which is 7-day point prevalence, as opposed to a continuous abstinence measure. Third, we used hypothetical instead of real monetary rewards in estimating delay discounting rates. Nevertheless, previous research has found comparable results from delay discounting procedures when real and hypothetical monetary rewards were used (Johnson & Bickel, 2002; Lagorio & Madden, 2005; Madden, Begotka, Raiff, & Kasten, 2003; Madden et al., 2004). Fourth, the sample was made of particularly moderate dependent group of smokers. Future studies should explore whether these results may be extended to smokers with lower or greater nicotine dependence.

Despite these limitations, the present study enhances the understanding of the relationship between delay discounting and smoking behavior. Although delay discounting did not change at short term after smoking cessation, it did decrease over time in those smokers who are abstinent at 12-month follow-up. The present results could have good generalizability since there were no significant differences in any baseline variable between the subjects excluded from the study

and those who were included. This finding supports the notion that delay discounting is mainly a state variable that may increase due to cigarette use but can also be reversed with the achievement of long term cigarette abstinence.

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Contributors

Secades-Villa R designed the study. García-Rodríguez O and Weidberg S managed the literature searches and wrote the first draft of the manuscript. Yoon JH conducted the statistical analyses. All authors contributed to and have approved the final manuscript.

Conflict of interest

No conflict declared.

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3.3. Artículo 3

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Resumen: Delay discounting and depressive symptomatology have strong connections with smoking. However, few studies have examined interactions across delay discounting, depressive symptoms, and smoking status. The primary goal of this secondary analysis was to assess the inter-relations across these three variables among treatment-seeking smokers. Delay discounting and depressive symptoms were assessed in 95 smokers enrolled in a clinical trial for smoking cessation at intake and 6-month follow-up. Participants with and without depressive symptoms did not differ in their discounting rates neither at intake nor at 6-month follow-up. However, delay discounting was significantly lower among abstainers at 6-month follow-up and, changes in discounting associated with smoking status were more pronounced among participants with depressive symptoms. These results clarify the relationship between delay discounting and depressive symptoms among current and former smokers and suggest that decreases in delay discounting associated with smoking abstinence are significantly greater among individuals with depressive symptoms versus those who do not have depressive symptomatology.

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Interaction of Depressive Symptoms and Smoking Abstinence on Delay Discounting Rates

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Delay discounting and depressive symptomatology have strong connections with smoking. However, few studies have examined interactions across delay discounting, depressive symptoms, and smoking status. The primary goal of this secondary analysis was to assess the interrelations across these 3 variables among treatment-seeking smokers. Delay discounting and depressive symptoms were assessed in 95 smokers enrolled in a clinical trial for smoking cessation at intake and 6-month follow-up. Participants with and without depressive symptoms did not differ in their discounting rates neither at intake nor at 6-month follow-up. However, delay discounting was significantly lower among abstainers at 6-month follow-up, and changes in discounting associated with smoking status were more pronounced among participants with depressive symptoms. These results clarify the relationship between delay discounting and depressive symptoms among current and former smokers and suggest that the association between smoking abstinence and lower delay discounting is significantly greater among individuals with depressive symptoms versus those who do not have depressive symptomatology.

Keywords: smoking, delay discounting, depression

Smoking is a major cause of premature mortality and preventable morbidity in the world, and up to one half of all the smokers will die from smoking-related diseases (World Health Organization, 2013). Despite public efforts to prevent smoking initiation and promote cessation, approximately 27% of the population in the European Union and 18% in United States are still current smokers (Agaku, King, Dube, & the Centers for Disease Control and Prevention, 2014; Gallus et al., 2014).

Previous studies have observed robust and reliable associations between depressive symptomatology and smoking. For example, smoking and depressive symptoms frequently co-occur (Chaiton, Cohen, O'Loughlin, & Rehm, 2009; Horton & Loukas, 2013; Mendelsohn, 2012). Additionally, smokers report more depressive symptoms compared with nonsmokers (Flensburg-Madsen et al., 2011; Khaled et al., 2012), and depressive symptomatology increases the likelihood of progressing into more intense smoking

(Audrain-McGovern, Lerman, Wileyto, Rodriguez, & Shields, 2004; Rohde, Kahler, Lewinsohn, & Brown, 2004). Finally, higher levels of both past and current depression are associated with lower abstinence rates following smoking treatment (Hitsman et al., 2013; Sonne et al., 2010).

Delay discounting describes the devaluation of a reinforcer as a function of increasing delay to its receipt (Odum, 2011; Reynolds, 2006). Relatively higher rates of discounting are considered an index of impulsivity (MacKillop et al., 2011). Considerable research has assessed delay discounting among individuals with substance use disorders, with the majority of work conducted in cigarette smokers (Yi, Mitchell, & Bickel, 2010). For example, cigarette smokers exhibit greater discounting compared with matched, nonsmoker controls (Baker, Johnson, & Bickel, 2003; Bickel, Odum, & Madden, 1999; Friedel, Dehart, Madden, & Odum, 2014; García-Rodríguez, Secades-Villa, Weidberg, & Yoon, 2013; Odum, Madden, & Bickel, 2002; Reynolds, Richards, Horn, & Karraker, 2004), and greater delay discounting is positively associated with severity of nicotine dependence (Amlung & MacKillop, 2014; Ohmura, Takahashi, & Kitamura, 2005; Rezvanfarad, Ekhtiari, Mokri, Djavid, & Kaviani, 2010; Sweitzer, Donny, Dierker, Flory, & Manuck, 2008). Additionally, several clinical studies have shown that greater baseline discounting among smokers predicted lower abstinence rates and increased risk of smoking relapse (Krishnan-Sarin et al., 2007; Sheffer et al., 2012; Yoon et al., 2007). Finally, delay discounting increases following acute smoking deprivation (Mitchell, 2004; Yi & Landes, 2012), while it decreases after a period of prolonged smoking abstinence (Secades-Villa, Weidberg, García-Rodríguez, Fernández-Hermida, & Yoon, 2014).

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Although both depressive symptoms and delay discounting have strong associations with smoking, few studies have examined the relationship between delay discounting and depressive symptomatology among smokers. Previous studies have found depression and smoking were independently related to greater discounting (Gatchalian, Yi, Bickel, Johnson, & Baker, 2004; Imhoff, Harris, Weiser, & Reynolds, 2014). Also, greater discounting was associated with higher depression symptoms (Imhoff et al., 2014; Yoon et al., 2007), and this relationship was even strengthened with greater nicotine dependence (Rezvanfard et al., 2010).

However, the results from the above studies must be interpreted with caution. First, relationships between delay discounting and depressive symptoms were often secondary findings and not the primary research focus of some of these studies (Rezvanfard et al., 2010; Yoon et al., 2007). Second, several studies were conducted in special populations such as students (Imhoff et al., 2014; Rezvanfard et al., 2010) and pregnant women (Yoon et al., 2007), which may limit the generalizability of these findings. Third, none of the prior studies assessed whether quitting smoking impacts the association between delay discounting and depressive symptomatology.

Therefore, the purpose of the current study was to assess the main and interactive effects of depressive symptoms and changes in smoking status on delay discounting rates among adult smokers who received a treatment for smoking cessation.

Method

Participants and Procedure

Participants were 116 patients who enrolled in a clinical trial for smoking cessation. The purpose of that trial was to compare 6-weeks cognitive-behavioral treatment (CBT) alone or combined with contingency management (CM; CBT + CM). Treatment protocols and procedures are described elsewhere (Secades-Villa, García-Rodríguez, López-Núñez, Alonso-Pérez, & Fernández-Hermida, 2014). As the purpose of the present study did not involve assessing the effect of treatment condition, participants in both groups were combined. Of these 116 participants, 14 were lost to 6-month follow-up and 7 were excluded because they presented nonsystematic delay discounting data (Johnson &

Bickel, 2008), so the final secondary analyses were conducted on these 95 participants, of which 30 received CBT and 65 received CBT + CM. Inclusion criteria consisted of being ≥ 18 years of age; report smoking ≥ 10 cigarettes per day for the last year; and meet diagnostic criteria for nicotine dependence according to the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., text rev.; *DSM-IV-TR*; American Psychiatric Association, 2000). Participants were excluded if they had a current diagnosis of a serious psychiatric disorder, other substance use disorder besides nicotine dependence, or if they were receiving any other smoking cessation treatment. Participants' characteristics are presented in Table 1. This study was approved by the Institutional Review Board of the University of Oviedo, and informed consent was obtained from all participants prior to study initiation.

Assessments and Dependent Measures

Participants' sociodemographic and smoking-related characteristics were assessed during the ~ 1.5 -hr intake session. The Fagerström Test for Nicotine Dependence (FTND; Heatherton, Kozlowski, Frecker, & Fagerström, 1991) was used to assess nicotine dependence in addition to the Structured Clinical Interview (SCID-I) of the *DSM-IV-TR* (American Psychiatric Association, 2000).

The Beck's Depression Inventory, Second Edition (BDI-II; Beck, Steer, & Brown, 1996) was used to assess depressive symptoms at intake and at 6-month follow-up. Scores ranging from 0 to 13 represent normal to minimal depression. Thus, participants with a score > 14 were classified as having depressive symptomatology in the current study.

Smoking status was also assessed at intake to corroborate current smoking and at 6-month follow-up. Biochemical measures of cigarette smoking included breath carbon monoxide (CO; Micro Smokerlyzer, Bedfont Scientific, Rochester, United Kingdom) and quantitative urinary cotinine (BS-120 chemistry analyzer, Shenzhen Mindray Bio-medical Electronics, Shenzhen, China). Smoking abstinence at 6-month follow-up was defined as self-reported smoking abstinence in the last 7 days (Cavollo et al., 2007), presenting a breath CO level ≤ 4 ppm (Perkins, Karelitz, & Jao, 2013), and presenting a urinary cotinine sample of < 80 ng/ml. If

Table 1
Participant Sociodemographic Characteristics at Intake

	With depressive symptoms (n = 30)	Without depressive symptoms (n = 65)	p value
Gender (% women)	76.7	60	.176
Age (years) ^a	41.53 \pm 13.26	47.23 \pm 13.40	.056
Marital status (% married)	36.7	49.2	.363
Cigarettes per day ^a	21.40 \pm 8.59	20.74 \pm 8.94	.735
Years of regular smoking ^a	24.17 \pm 12.50	27.32 \pm 13.88	.285
FTND ^a	5.47 \pm 2.36	5.5 \pm 1.83	.791
CO (ppm) ^a	13.33 \pm 4.88	14.83 \pm 6.58	.269
Cotinine (ng/ml) ^a	2,354 \pm 1,389	2,287 \pm 1,121	.812
BDI-II ^a	21.17 \pm 6.58	5.46 \pm 3.39	<.01

Note. FTND = Fagerström Test for Nicotine Dependence; CO = carbon monoxide; ppm = parts per million; ng/ml = nanogram/milliliter; BDI-II = Beck Depression Inventory: Second Edition.
^a Means \pm SD.

a participant did not satisfy any one of these criteria, he or she was classified as a smoker.

The delay discounting task was presented to participants at intake and at 6-month follow-up via a laptop running the Windows operating system. The task took approximately 10 min to complete for each participant. Participants were provided instructions on how to interact with the delay discounting program and informed that they would not receive any of the monetary amounts presented, but they were asked to respond as if the choices were real. Details about the delay discounting task are described elsewhere (García-Rodríguez et al., 2013). Participants were presented with repeated choices between €1,000 after a fixed delay versus various amounts of money ranging from €5 to €955 available immediately using an adjusting-amounts procedure (Holt, Green, & Myerson, 2012). Based on the participant's responding, the value of the immediate monetary option ranged from €5 to €1,000 in €5 increments and was adjusted via a titrating procedure. This procedure yields an indifference point, in which the value the immediate amount and the delayed €1,000 is deemed equal, and this process is repeated at several delays (Rachlin, Raineri, & Cross, 1991). The delays values were 1 day, 1 week, 1 month, 6 months, 1 year, 5 years, and 25 years. The delays were presented in an ascending order for all the participants.

Data Analysis

In order to assess k values for each participant, individual indifference points were fitted to the hyperbolic equation described by Mazur (1987):

$$V = A/(1 + kD) \quad (1)$$

The equation (1) shows how the value (V) of a reward of certain amount (A) is discounted as a function of delay (D) to its delivery (Mazur, 1987). The free parameter k describes the rate of discounting, with higher k values showing greater discounting and more impulsive choices selected. The hyperbolic model was fitted to each subject's delay discounting data (i.e., indifference points) with nonlinear regression (SAS, PROC NLIN). Goodness of fit was evaluated on the basis of model R^2 values. As the distribution of k values was skewed, analyses were performed on log-transformed k values. Nonetheless, descriptive delay discounting data are presented in a nonlogged k format (i.e., geometric mean k values). Nonsystematic delay discounting data were identified and eliminated using the algorithm described by Johnson and Bickel (Johnson & Bickel, 2008).

Analyses were also conducted using the Area Under the Curve (AUC) as a secondary dependent measure. Myerson, Green, and Warusawitharana (2001) proposed AUC as a theoretically neutral measure of delay discounting that avoids assumptions of any particular discounting model (Odum, 2011; Odum & Rainaud, 2003).

Independent-samples t tests were conducted to assess if baseline $\log k$ and AUC values differed between participants with and without depressive symptoms at both intake and 6-month follow-up, and as a function of smoking status at 6-month follow-up. A two-way between-groups analysis of variance (ANOVA; after Levene's test for homogeneity of variances) was performed to assess the main and interactive effects of depressive symptomatol-

ogy at 6-month follow-up (participants with vs. without depression symptoms) and smoking status (smokers vs. abstainers) on participants' delay discounting rates at 6-month follow-up. Additionally, independent-samples t tests were performed to reassess significant interactive effects of depressive symptomatology and smoking status. Effect sizes of principal comparisons were calculated using partial eta squared (η_p^2) statistic. Confidence level was 95%, and the statistical package was the SPSS (V15; SPSS, Chicago, IL).

Results

Equation (1) provided a good fit to participants' delay discounting data. The median R^2 was .92. Approximately 73% of the R^2 scores were greater than .80, and only 11% of them were lower than .30.

Delay Discounting and Depressive Symptomatology at Intake

Participants' geometric mean k and AUC values at intake were 0.0077 ($SE = 0.4393$) and .2261 ($SE = .0195$), respectively. Baseline delay discounting rates between participants with ($M_k = 0.0066$, $SE_k = 1.1442$; $M_{AUC} = .2296$, $SE_{AUC} = .0348$) and without depressive symptoms ($M_k = 0.0082$, $SE_k = 0.3699$; $M_{AUC} = .2244$, $SD_{AUC} = .0238$) at intake were not significantly different, $t_{\log k}(93) = 0.334$, $p = .739$; $t_{AUC}(93) = -0.122$, $p = .903$.

Baseline delay discounting between participants with ($M_k = 0.0165$, $SE_k = 0.3433$; $M_{AUC} = .1697$, $SE_{AUC} = .0382$) and without depressive symptoms ($M_k = 0.0063$, $SE_k = 0.5495$; $M_{AUC} = .2411$, $SE_{AUC} = .0224$) at 6-month follow-up was not significantly different, $t_{\log k}(93) = -1.308$, $p = .194$; $t_{AUC}(93) = 1.499$, $p = .137$.

Smokers ($M_k = 0.0102$, $SE_k = 0.1414$; $M_{AUC} = .1976$, $SE_{AUC} = .0227$) and abstainers ($M_k = 0.0056$, $SE_k = 0.9503$; $M_{AUC} = .2603$, $SD_{AUC} = .0328$) at 6-month follow-up did not differ in their baseline delay discounting rates, $t_{\log k}(93) = -1.010$, $p = .315$; $t_{AUC}(93) = 1.607$, $p = .112$.

Delay Discounting and Depressive Symptomatology at 6-month Follow-Up

Approximately 45% of the participants were abstinent at 6-month follow-up (6 participants with depressive symptoms and 37 without depressive symptoms), and 55% of the participants were smoking at 6-month follow-up (14 participants with depressive symptoms and 38 without depressive symptoms). Mean BDI-II scores among abstainers and smokers at 6-month follow-up were 6.14 ($SD = 6.35$) and 8.77 ($SD = 8.45$), respectively. Levene's test for homogeneity of variances for $\log k$ and AUC values showed no significant differences in the variances of both parameters across the four groups (abstainers and smokers with and without depressive symptoms). Results from two-way between-groups ANOVA revealed a significant main effect of smoking status in delay discounting rates at 6-months follow-up, $F_{\log k}(1, 91) = 5.608$, $p = .020$, $\eta_p^2 = .058$; $F_{AUC}(1, 91) = 9.091$, $p = < .01$, $\eta_p^2 = .091$. Specifically, abstainers ($M_k = 0.0032$, $SE_k = 0.0253$; $M_{AUC} = .2805$, $SE_{AUC} = .0323$) exhibited significantly lower delay discounting than smokers ($M_k = 0.0051$,

$SE_k = 0.0534$; $M_{AUC} = .2197$, $SD_{AUC} = .0237$). There was no significant effect of depressive symptoms in delay discounting rates, $F_{\log k}(1, 91) = 0.232$, $p = .631$; $F_{AUC}(1, 91) = 1.193$, $p = .278$. However, there was a significant Depressive Symptoms \times Smoking Status interaction effect, $F_{\log k}(1, 91) = 6.581$, $p = .012$, $\eta_p^2 = .067$; $F_{AUC}(1, 91) = 7.811$, $p = < .01$, $\eta_p^2 = .079$. Specifically, among those with depressive symptoms, delay discounting rates were significantly lower among abstainers ($M_k = 0.0007$, $SE_k = 0.0269$; $M_{AUC} = .4470$, $SE_{AUC} = .1371$) compared with smokers ($M_k = 0.0125$, $SE_k = 0.1936$; $M_{AUC} = .1577$, $SD_{AUC} = .0479$), $t_{\log k}(18) = -2.109$, $p = .049$; $t_{AUC}(18) = 2.538$, $p = .021$. In contrast, no significant differences were observed between abstainers ($M_k = 0.0041$, $SE_k = 0.0291$; $M_{AUC} = .2535$, $SD_{AUC} = .0290$) and smokers ($M_k = 0.0037$, $SE_k = 0.0101$; $M_{AUC} = .2425$, $SE_{AUC} = .0266$), $t_{\log k}(73) = 0.252$, $p = .801$; $t_{AUC}(73) = 0.279$, $p = .781$, without depressive symptoms. Figure 1 shows the geometric mean k values for both abstainers and smokers with or without depressive symptoms. Higher values along the y-axis indicate greater delay discounting.

Discussion

To our knowledge, this is the first study to assess the interrelations of smoking status, depression symptomatology, and delay discounting among adult seeking-treatment smokers. There were three major findings: (a) participants with and without depressive symptoms did not differ in their delay discounting rates neither at intake nor at 6-month follow-up; (b) delay discounting rates were significantly lower among abstainers at 6-month follow-up; and (c) depressive symptoms appear to impact the association between delay discounting and smoking status, such that the relationship between smoking abstinence and low delay discounting is greater among participants with depressive symptoms.

Contrasting with previous studies (Gatchalian et al., 2004; Imhoff et al., 2014; Yoon et al., 2007) but in line with results from others (Dennhardt & Murphy, 2011; Dombrovski et al., 2011; Gonzalez, Reynolds, & Skewes, 2011), we found no relationship

between delay discounting and depressive symptoms at both intake and 6-month follow-up. The different types of populations and measures of depressive symptoms employed in the studies may explain this divergent result. Specifically, it is possible that elevated delay discounting may only be found among individuals who meet diagnostic criteria for major depressive disorder (Takahashi et al., 2008), given that executive functioning, which affects delay discounting, has shown to be unimpaired among partly remitted depressed participants when compared with healthy controls (Westheide et al., 2007).

Delay discounting rates were significantly lower among abstainers than smokers at 6-month follow-up. A previous study also showed that changes in smoking status were associated with changes in delay discounting rates (Secades-Villa, Weidberg et al., 2014). This finding may be the result of increases in alternative positive reinforcement associated with lifestyle changes that occur with smoking abstinence (Jang et al., 2012; Nagaya, Yoshida, Takahashi, & Kawai, 2007). Moreover, neurobiological modifications (Bechara, 2005; Bickel et al., 2007) or reversal of processing bias (Munafò, Mogg, Roberts, Bradley, & Murphy, 2003) can also account for this result. Regardless of which mechanism is responsible, the current finding further supports the notion that delay discounting reductions may be associated with longer-term abstinence.

The relationship between smoking status and delay discounting was stronger among participants with depressive symptoms than those without such symptoms, a finding not previously documented to our knowledge. There are a few possible factors that might help elucidate these findings. First, being depressed (Smoski et al., 2008) and abstinent from smoking (Goto, Takahashi, Nishimura, & Ida, 2009) are both positively related to high risk aversion (tendency to evaluate a prospect with positive probabilistic outcomes as having a value lower than its expected value; Glöckner & Hilbig, 2012), and there is evidence of an inverse relationship between risk aversion and delay discounting (Buelow, Okdie, & Blaine, 2013). Second, the effects of smoking abstinence on the adoption of healthy lifestyles inversely related to delay discounting (e.g., attempt to lose weight, increase exercise, reduce fat intake, or increase fiber intake; Melanko & Larkin, 2013) could be stronger among individuals with depressive symptoms (Green & Pope, 2000). These effects may be heightened among individuals with depressive symptoms due to low rates of response-contingent positive reinforcement (Carvalho & Hopko, 2011), which may motivate them to engage in behavioral changes that would increase reinforcement rates. Third, a previous study showed that individuals with depressive symptoms who successfully quit smoking also significantly reduced their frequency of alcohol use (Prochaska et al., 2008), which is positively related with reductions in delay discounting rates (Takahashi, Ohmura, Oono, & Radford, 2009). Finally, depressive smokers show enhanced attentional bias to smoking cues (Kushmir et al., 2013), and individuals with this bias have fewer problems reducing cigarette craving (Westbrook et al., 2013). Abstinence from smoking among participants with depressive symptoms (Bradstreet et al., 2014) reduces craving, which may lead in turn to decreased delay discounting (Heinz, Peters, Boden, & Bonn-Miller, 2013). This study does not address whether all these factors actually contribute to explain the moderating effect of depressive symptomatology on the relationship between delay discounting and changes in smok-

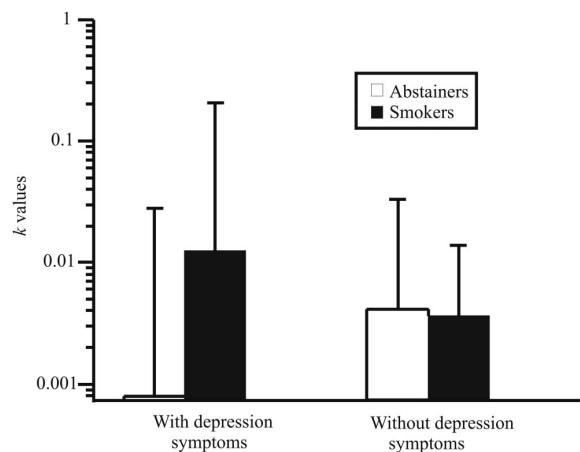


Figure 1. Geometric mean k values for abstainers and smokers with or without depression symptoms at 6-month follow-up. Error bars represent the standard error.

ing status. This suggests the importance for future studies to support their feasibility to explain the present findings.

The greater discounting rates observed among participants with depressive symptoms who smoked at 6-month follow-up suggests that these individuals' smoking may be less influenced by the longer-term consequences of smoking and more by the immediate reinforcing consequences of smoking. To the extent that this is true, treatments providing alternative immediate reinforcement for smoking abstinence, such as CM, may be particularly suited for smokers with depressive symptoms. For example, previous studies have shown that CM can improve abstinence rates among those with comorbid substance use disorder and depression (Drebing et al., 2005; Gonzalez, Feingold, Oliveto, Gonsalves, & Kosten, 2003).

Several limitations of the present study should be noted. First, delay discounting was assessed using hypothetical monetary rewards rather than real monetary consequences. However, previous studies comparing hypothetical versus real monetary rewards have shown no systematic differences in delay discounting (Dixon, Lik, Green, & Myerson, 2013; Johnson & Bickel, 2002; Lagorio & Madden, 2005; Madden, Begotka, Raiff, & Kasten, 2003). Second, diagnostic interviews (such as the SCID-II) were not used to assess depression. However, the BDI is a valid and reliable clinical tool used to screen for depression, psychometrically supported, and widely used in community settings (Hides, Samet, & Lubman, 2010). Third, the relatively small number of participants with depressive symptoms and the fact that this was not a clinical sample of depressed smokers may limit the generalizability of the findings.

Even with these limitations, the present study provides novel insight into the association between delay discounting and depression among persistent and former smokers. Our results suggest that the association between smoking abstinence and lower delay discounting is enhanced among individuals with depressive symptoms.

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4. ESTUDIO COMPLEMENTARIO

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Resumen: Increasing evidence suggests that delay discounting may change following effective interventions. Nonetheless, previous studies that assessed the effect of contingency management (CM) on delay discounting are scarce and their results mixed. The current study assessed if contingency management (CM) in conjunction with a cognitive-behavioral treatment (CBT) for smoking cessation was associated with changes in delay discounting at end-of-treatment and at 6-month follow-up compared to CBT alone. One hundred and sixteen treatment-seeking smokers were randomly assigned to either CM + CBT ($n = 69$) or CBT alone ($n = 47$). Participants completed delay discounting assessments at the intake, at end-of-treatment and at 6-month follow-up. We evaluated CM's effect on discounting with parametric and nonparametric methods. Most results failed to show a statistical reduction in discounting attributable to CM. Although smoking abstinence did not affect changes in delay discounting, delay discounting tended to decrease more among abstainers compared to those participants who did not quit. The current results suggest that CM intervention is not robustly associated with delay discounting changes. Future studies should address treatments that may potentially change delay discounting.

Abstract

Increasing evidence suggests that delay discounting may change following effective interventions. Nonetheless, previous studies that assessed the effect of contingency management (CM) on delay discounting are scarce and their results mixed. The current study assessed if contingency management (CM) in conjunction with a cognitive-behavioral treatment (CBT) for smoking cessation was associated with changes in delay discounting at end-of-treatment and at 6-month follow-up compared to CBT alone. One hundred and sixteen treatment-seeking smokers were randomly assigned to either CM + CBT ($n = 69$) or CBT alone ($n = 47$). Participants completed delay discounting assessments at the intake, at end-of-treatment and at 6-month follow-up. We evaluated CM's effect on discounting with parametric and nonparametric methods. Most results failed to show a statistical reduction in discounting attributable to CM. Smoking abstinence did not affect changes in delay discounting. The current results suggest that CM intervention is not robustly associated with delay discounting changes. Future studies should address treatments that may potentially change delay discounting.

Keywords: Delay discounting, smoking, contingency management, cognitive-behavioral treatment.

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Contingency Management and Delay Discounting among Patients Receiving Smoking Cessation Treatment

Delay discounting has substantial relevance for understanding behavioral processes associated with a wide range of problematic behaviors, including substance use disorders (SUDs) (*for reviews see* Bickel, Koffarnus, Moody, y Wilson, 2014; Bickel y Marsch, 2001; Reynolds, 2006). Greater discounting has been consistently observed among those with SUDs compared to matched controls across a wide variety of drugs of abuse (Baker, Johnson, y Bickel, 2003; Coffey, Gudleski, Saladin, y Brady, 2003; Dom, D'Haene, Hulstijn, y Sabbe, 2006; García-Rodríguez, Secades-Villa, Weidberg, y Yoon, 2013; Heil, Johnson, Higgins, y Bickel, 2006; Hoffman et al., 2006; Kirby y Petry, 2004; Odum, Madden, y Bickel, 2002; Petry, 2001). Greater delay discounting (or a similar construct of delay gratification) is associated with earlier drug use onset (Audrain-McGovern et al., 2009; Ayduk et al., 2000), greater addiction severity (Cheng, Lu, Han, Gonzalez-Vallejo, y Sui, 2012; MacKillop et al., 2010; Ohmura, Takahashi, y Kitamura, 2005; Reynolds, 2004; Rezvanfard, Ekhtiari, Mokri, Djavid, y Kaviani, 2010), and lower rates of abstinence in both laboratory models (Dallery y Raiff, 2007; Mueller et al., 2009) and clinical trials (MacKillop y Kahler, 2009; Sheffer et al., 2012; Stanger et al., 2012; Yoon et al., 2007).

Some correlational (Baker, et al., 2003; Takahashi, Furukawa, Miyakawa, Maesato, y Higuchi, 2007) and clinical studies (Black y Rosen, 2011) have observed that delay discounting is relatively stable over time among those with SUDs . Nonetheless, there is increasing evidence suggesting that delay discounting may change under different pharmacological or environmental conditions (Dallery y Raiff, 2007; Koffarnus, Jarmolowicz, Mueller, y Bickel, 2013). For instance, increases in delay d

discounting have been observed following acute administration of alcohol among social drinkers (Reynolds, Richards, y de Wit, 2006). Likewise, increased discounting has been observed following acute deprivation of their drug of choice among cigarette smokers (Ashare y Hawk, 2012; Field, Santarcangelo, Sumnall, Goudie, y Cole, 2006; Mitchell, 2004; Yi y Landes, 2012) and opioid-dependent individuals (Giordano et al., 2002). Discounting rates also increased among pathological gamblers in gambling contexts (Dixon y Holton, 2009; Dixon, Jacobs, y Sanders, 2006).

Relatively few studies have examined the effect of clinical interventions on delay discounting among individuals with SUDs. Among those completed, decreased delay discounting has been observed following a working memory training procedure for stimulant-dependent individuals (Bickel, Yi, Landes, Hill, y Baxter, 2011) and a money-management intervention for cocaine- and/or alcohol-dependent individuals (Black y Rosen, 2011). The effects of contingency management (CM) on delay discounting have been mixed. Two studies showed that CM led to significant reductions in delay discounting rates among smokers (Yi et al., 2008) and opioid-dependent individuals receiving multimodal treatments of which CM was a part of each (Landes, Christensen, y Bickel, 2012), whereas no changes in delay discounting were observed among marijuana-dependent individuals receiving CM treatment (Peters, Petry, LaPaglia, Reynolds, y Carroll, 2013).

Taken together, these results suggest that delay discounting may change in response to effective CM treatment, but the limited number of studies makes necessary further research to confirm these findings (Bickel, et al., 2014). Furthermore, a common limitation of previous research is the lack of statistical control of the interaction effect

between treatment condition and abstinence from drug use (Landes, et al., 2012; Peters, et al., 2013; Yi, et al., 2008), leading to potential confounding effects. Also, whether changes in delay discounting persisted following termination of treatment is unknown as these studies only report end-of-treatment results.

The present study addresses this gap in the literature investigating whether CM in conjunction with cognitive-behavioral treatment (CBT) for smoking cessation was associated with changes in delay discounting at end-of-treatment and at 6-month follow-up.

Method

Participants

Participants were 123 individuals seeking treatment for cigarette smoking at the Addictive Behaviors Clinic of the University of Oviedo (Spain). Inclusion criteria were as follows: being over 18 years old, smoking 10 or more cigarettes per day for the last year and meeting criteria for nicotine dependence according to the Diagnostic and Statistical Manual of Mental Disorders (4th ed., text rev.; DSM–IV–TR) (American Psychiatric Association, 2000). Participants were excluded if they were diagnosed with a current psychiatric disorder (including substance use disorder besides nicotine dependence) or if they were receiving any other smoking cessation treatment. This study was approved by the Institutional Review Board of the University of Oviedo, and informed consent was obtained from all participants prior to study initiation. Seven participants (3 assigned to the CM + CBT condition and 4 assigned to the CBT condition) were excluded because they presented nonsystematic delay discounting data (Johnson y Bickel, 2008).

Eligible participants were randomly assigned to either the CM + CBT condition ($n = 69$) or CBT condition ($n = 47$). Table 1 shows the counts of participants who supplied delay discounting data at all the assessments (intake, end-of-treatment and 6-month follow-up) as well as those who missed one or more of these assessments. There were no significant differences between conditions in any sociodemographic and smoking-related variables (Table 2) or delay discounting rates at the intake (Table 3).

Instruments and variables

Sociodemographic and smoking-related characteristics were assessed during the intake session, which lasted about 1 and a half hours. The Structured Clinical Interview (SCID-I) of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR) (American Psychiatric Association, 2000) was used to assess nicotine dependence.

The delay discounting task was presented to participants at intake, end-of-treatment and 6-month follow-up via a laptop running the Windows Operating system. The task took approximately 10 minutes to complete for each participant. Participants were instructed how to interact with the delay discounting program and informed that they would not receive any of the monetary amounts presented, but they had to respond as if the choices were real. Participants were presented with a choice between €1,000 (US\$ 1216.06) after a fixed delay versus various amounts of money available immediately using an adjusting-amounts procedure (Holt, Green, y Myerson, 2012). The delays values used were 1 day, 1 week, 1 month, 6 months, 1 year, 5 years and 25 years. The delays were presented in an ascending order for all the participants. The value of the immediate monetary option ranged from €5 (US\$ 6.08) to €1,000 (US\$ 1216.06) in €5(US\$ 6.08) increments and was adjusted via a titrating procedure that

honed in on the indifference point based on the participants' responses. The titration procedure took the lower and upper limit of possible values [(initial €/US\$0 and €1,000 (US\$ 1216.06)] and divided this total range by 2, 3 or 4 to obtain an interval value. The value of the immediate option was one interval value above or below the upper and lower limits. If the immediate value was outside €/US\$0 and €1,000 (US\$ 1216.06), another value was randomly chosen. New lower and upper limits were chosen based on the participant's response, adjusting the total range, and this titration procedure was repeated for each of the seven delays. Note that based on the possible values presented, the total values could occasionally increase if they chose an option outside of the total range. Once the total range was at or less than €40 (US\$ 48.21), the average of the upper and lower limits was taken as the indifference point, and the next delay was presented.

Smoking status was assessed at intake, end-of-treatment and 6-month follow-up. Participants provided a breath carbon monoxide (CO) using a Micro Smokerlyzer (Bedfont Scientific Ltd., Rochester, UK). A BS-120 chemistry analyzer (Shenzhen Mindray Bio-medical Electronics, Co., Ltd., Shenzhen, P. R. China) was also used to determine quantitative urine cotinine levels. Self-reported smoking abstinence at the end-of-treatment and at 6-month follow-up was validated by presenting a breath CO level \leq 4 ppm (Perkins, Karelitz, y Jao, 2013) and a cotinine test $<$ 80 ng/ml. Agreement between the three measures was required to be categorized as abstinent. Participants were considered as smokers when they missed an assessment.

Treatment conditions

CBT.

CBT consisted of a 6-week intervention described in previous studies (Becoña y Vázquez, 1997; Secades-Villa, Alonso-Perez, García-Rodríguez, y Fernández-Hermida, 2009; Secades-Villa, García-Rodríguez, López-Núñez, Alonso-Pérez, y Fernández-Hermida, 2014). CBT was implemented in group-based sessions of six patients. Each weekly session lasted about 1 hour. The components of the CBT were highly structured and included: information about tobacco, a behavioral contract, self-monitoring and graphical representation of cigarette smoking, nicotine fading, stimulus control, strategies for controlling nicotine withdrawal symptoms, physiological feedback consumption, training in alternative behaviors, social reinforcement of objectives completion and abstinence, and relapse prevention strategies. CO and cotinine specimens were collected twice a week. One of the measures coincided with the weekly CBT session and the other was scheduled midweek between sessions.

CM + CBT.

The CM + CBT condition was similar to the CBT condition, but with the addition of CM. Participants were randomly assigned to two types of CM procedures: CM for smoking abstinence or CM for shaping abstinence. The CM for smoking abstinence included a voucher program in which nicotine abstinence was reinforced on an escalating schedule of reinforcement with a reset contingency. Points were earned for specimens testing negative for cotinine (< 80 ng/ml) from the fifth session forward (once the patients were required to be completely abstinent) (Secades-Villa, García-Rodríguez, et al., 2014). The CM for shaping abstinence included a voucher program in which progressive reductions in cotinine (with abstinence also as final target) were reinforced according to an individualized percentile schedule. Points were earned for

specimens that met the reduction criteria from the first session forward (Lamb, Kirby, Morral, Galbicka, y Iguchi, 2010; Secades-Villa, López-Núñez, Pericot-Valverde, Alonso-Pérez, y García - Rodríguez, 2014). Points were worth the equivalent to 1€ (US\$ 1.36) each. The maximum amount that patients could earn in both procedures was 300€. Points were exchangeable for vouchers with a variety of uses, including leisure activities, cinema, theater, museums, sport events, gyms, adventure sports, meals in restaurants, training, purchases in department stores, and spa and beauty services (Secades-Villa, García-Rodríguez, et al., 2014). In both procedures, participants were encouraged by therapists to consider spending their vouchers on goods and services that promote a healthier lifestyle. Since no significant differences in abstinence rates between the two CM conditions were observed (Secades-Villa, et al., 2014), data from the two conditions were combined for the current study.

Data Analysis

We summarized the indifference points from each discounting using the area under the curve – AUC. Myerson, Green y Warusawitharana (2001) proposed AUC as an atheoretic discounting measure that avoids assumptions of any particular discounting model (Odum, 2011; Odum y Rainaud, 2003). AUC takes values from 1 (no discounting) to 0 (maximum discounting).

To measure within-individual change in discounting between intake and either end-of-treatment or 6-month follow-up, we took the difference. In particular, AUC change at end-of-treatment was defined as intake AUC minus end-of-treatment AUC, and AUC change at 6-month follow-up as 6-month follow-up AUC minus intake AUC. Computed as such, a negative difference indicates a decrease from intake discounting in

AUC. We focus our analyses on the differences from intake discounting, and refer to these as d_{AUC} .

We analyzed the d_{AUC} data in an analysis of variance (ANOVA) context having CM as a between-group factor, and both assessment period and CM \times assessment period as within-group factors. Using the Bayesian information criterion, we chose a compound symmetric covariance structure (equivalent to a first-order autoregressive structure) over an unstructured covariance. As recommended by Littell, Milliken, Stroup, Wolfinger, y Schabenberger (2006), Kenward-Roger method was used in order to estimate the error degrees of freedom.

Although a population may experience a mean significant decrease on delay discounting over time, individuals may deviate from this pattern by either not shifting at all, or shifting in the opposite direction. In order to assess whether each individual showed a statistically significant change in his or her delay discounting from one assessment period to another, we used the sign rank test described in Hadden (2012). This test uses the differences of indifference points paired on the delay from which they came while assuming no mathematical model of discounting. We tested whether each individual statistically changed from intake to both end-of-treatment and 6-month follow-up. We then used χ^2 tests to compare the CM groups for differences in the proportions of those statistically changing.

χ^2 tests were also performed to assess whether abstinence rates at both end-of-treatment and 6-month follow-up differed as a function of treatment condition. Finally, to assess if changes in discounting were associated to both the receipt of CM and

smoking status, an ANOVA on discounting change measures was conducted accounting for CM, smoking status and their interaction.

Primary analyses were conducted in SAS/STAT software, Version 9.3, SAS System for Windows (SAS Institute Inc., Cary, NC, USA), with linear mixed models fitted in the MIXED procedure. Confidence level was 95%.

Results

Delay Discounting Changes and Treatment Conditions

For each CM group and assessment period, Tables 3 provides summary statistics of the delay discounting measures, and Table 4 the estimated change from intake discounting (d). Averaging over end-of-treatment and 6-month follow-up, change from intake discounting (d) did not significantly differ between the two treatment conditions [$F_{AUC}(1, 112) = 1.25, p = .267$]; nor was there evidence of a CM \times assessment period interaction [$F_{AUC}(1, 105) = 1.04, p = .310$]. However, the CM + CBT group evidenced decreased discounting at the end-of-treatment ($p_{AUC} = .021$), and the discounting decrease of this group at 6-month follow-up approached significance ($p_{AUC} = .097$); however CBT participants did not decrease their discounting at each assessment period (all $p \geq .538$). Averaged the change over the two time periods, change from intake discounting was significantly decreased in the CM + CBT group [$t_{AUC}(110) = 2.21, p = .029$], but statistically unchanged in the CBT group [$t_{AUC}(114) = 0.35, p = .726$].

Distributions of Individually-Based Changes in Delay Discounting

Figures 1 and 2 show the proportions of participants who statistically decreased, experienced no significant change, or significantly increased their delay discounting

rates as a function of treatment condition at the end-of-treatment and at 6-month follow-up, respectively. There were no significant differences between the two treatment conditions in these proportions either at end-of-treatment [$\chi^2_{(2)} = 0.090, p = .956$] or at 6-month follow-up [$\chi^2_{(2)} = 1.053, p = .591$].

Please insert Figures 1 and 2 here

The estimated change from intake AUC to end-of-treatment by that at 6-month follow-up is plotted in Figure 3. The rank correlation between these two assessment periods were 0.61 ($p < .001, n = 39$) for the CBT group and 0.62 ($p < .001, n = 65$) for the CM + CBT group.

Please insert Figure 3 here

Delay Discounting and Abstinence Outcomes

Eighty-one percent of the participants were abstinent at the end-of-treatment. Participants in the CM + CBT condition achieved significantly higher abstinence rates (94.2 %) at this assessment period when compared to participants in the CBT condition (61.7 %), $\chi^2_{(1)} = 17.158, p = .001$. At 6-month follow-up, 39% of the participants maintained smoking abstinence. Abstinence rates at this assessment period did not significantly differ between participants in the CM + CBT condition (43.5%) and participants in the CBT condition (31.9%), $\chi^2_{(1)} = 1.125, p = .289$.

There was no significant effect of CM, smoking status or their interaction on delay discounting changes neither at end-of-treatment nor at 6-month follow-up (see Table 5 for the ANOVA results). Table 6 shows the estimated change from intake delay discounting rates among smokers and abstainers.

Discussion

The main purpose of the present study was to assess if CM added to a CBT intervention for smoking cessation was associated with changes in delay discounting at end-of-treatment and at 6-month follow-up. There are four noteworthy outcomes of the current study: 1) between-groups analyses showed no statistical differences between CBT and CM+CBT; 2) within-groups analyses showed that participants in the CM + CBT condition evidenced discounting decreases from intake to end-of-treatment, while the discounting decrease from intake to 6-month follow-up approached significance; participants in the CBT condition failed to show a statistical change across time; 3) distributions of individually-based changes in delay discounting were similar in both treatment conditions; and 4) smoking abstinence did not affect changes in delay discounting.

The vast majority of the analyses conducted showed that neither the CBT nor the CM + CBT interventions changed delay discounting from intake to both end-of-treatment and 6-month follow-up (with the exception of a significant decrease from intake to end-of-treatment in the CM + CBT group). Similar results were found in a previous work from Peters et al. (2013), who showed that marijuana dependents did not change their discounting after receiving a 12-week CM treatment, either alone or combined with CBT. Nonetheless, this previous study showed that participants who received CBT alone increased their discounting from intake to end-of-treatment, while in the present study participants who received CBT alone did not statistically change their discounting across time. It is possible that only specific treatments, such as those that directly target executive functioning (see Bickel et al., 2011) or psychopharmacological approaches that use cognitive enhancers (Bickel, Jarmolowicz,

Mueller, Koffarnus, y Gatchalian, 2012) are effective to reduce delay discounting. Future research should also assess which treatments or combination of treatment components is most effective in order to reduce impulsive decision making (Bickel et. al, 2012).

The within-groups reductions observed from intake to end-of-treatment among the CM + CBT condition may be primarily attributed to a more pronounced decrease in delay discounting at the individual level among participants who showed discounting reductions and received CM, compared to those participants who evidenced discounting reductions but did not receive CM. Factors such as immediate access to reinforcers for drug abstinence and the opportunity to consider spending vouchers on goods and services that promote a healthier lifestyle provided by CM (Chivers y Higgins, 2012; Higgins, Silverman, Sigmon, y Naito, 2012) may explain the significant decreases in delay discounting from intake levels among these specific individuals.

We found no effect of smoking status on delay discounting change neither at the end-of-treatment nor at 6-month follow-up. Previous results also showed that delay discounting remains stable as a function of initial (Dallery y Raiff, 2007; Yoon, Higgins, Bradstreet, Badger, y Thomas, 2009) and medium-term smoking abstinence (Yoon, et al., 2007), suggesting that a longer period of abstinence may be required to detect changes in delay discounting (Secades-Villa, Weidberg, García-Rodríguez, Fernández-Hermida, y Yoon, 2014). Nonetheless, participants who received CM achieved higher abstinence rates at the end-of-treatment, which may suggest that the more pronounced delay discounting reductions among some specific individuals who received CM, may be at least partially explained by abstinence from smoking.

Some limitations of the current study should be acknowledged when interpreting the results. First, delay discounting rates were assessed using hypothetical monetary rewards and one may question whether the present findings would be similar as for discounting of real rewards. Nonetheless, previous research has found that discounting rates from hypothetical and real rewards are highly and positively correlated (Baker, et al., 2003; Johnson y Bickel, 2002; Johnson, Bickel, y Baker, 2007; Lagorio y Madden, 2005; Madden, Begotka, Raiff, y Kastern, 2003; Madden et al., 2004). Second, this study defined abstinence by an agreement of self-reported and biological measures using a 7-day point prevalence at 6-month follow-up, instead of employing a measure of continuous abstinence. Third, the relatively small sample size may have made it difficult to find statistically significant outcomes in some of the analyses conducted.

Despite these limitations, our study shows that CM intervention did not appear to be robustly associated with delay discounting changes among a sample of treatment seeking adult smokers. Given the scarcity of studies that assessed whether any intervention, including CM, is related with delay discounting changes, more will be needed to confirm the present findings.

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Table 1

Counts of participants who supplied delay discounting data for each group and assessment

	Intake only	Intake + end-of-treatment	Intake + 6-month follow-up	All three assessments	Total
CBT	1	6	1	39	47
CBT + CM	0	4	0	65	69
Total	1 ^a	10	1	104	116

Note. CBT = cognitive-behavioral treatment; CM = contingency management.

^a = Data from this participant is included in analyses that relate to inference on intake-only measures.

Table 2*Participants' characteristics at intake*

	CBT (n = 47)	CM + CBT (n = 69)	p value
Age (years) ^a	47.53 ± 11.27	43.36 ± 13.78	.077
Gender (% women)	55.3	66.7	.298
Marital status (% married)	55.3	42	.492
Cigarettes per day ^a	23.66 ± 9.41	20.52 ± 8.52	.064
Years of regular smoking ^a	27.48 ± 10.95	25.18 ± 13.40	.336
CO (ppm) ^a	16 ± 6.86	14.51 ± 5.99	.217
Cotinine (ng/ml) ^a	2,267.8 ± 1,110	2,424 ± 1,272	.538

Note. CBT = cognitive-behavioral treatment; CM = contingency management; CO = carbon monoxide; ppm = parts per million; ng/ml = nanogram/milliliter.

^a = Means ± SD

Table 3

*Summary Statistics of AUC for Each Treatment Condition
and Assessment Period*

Treatment condition	Assessment period	N	AUC	
			Mean	Std Dev.
CBT	Intake ^a	47	0.18	0.15
	Intake	46	0.19	0.15
	End-of-treatment	45	0.19	0.19
	6-month follow-up	40	0.19	0.15
CM + CBT	Intake	69	0.23	0.18
	End-of-treatment	69	0.28	0.22
	6-month follow-up	65	0.26	0.20

Note. CBT = cognitive-behavioral treatment;

CM = contingency management; AUC = Area Under the Curve.

^aData including a participant who complete intake assessment only

Table 4*Estimated Mean Change from Intake AUC with Comparisons of CM Groups Added*

		AUC		
Assessment period	Treatment condition	Mean Change ^a	95% CI ^a	t-statistic yp-value
End-of-treatment	CBT	-0.0	(-5.7, +5.6)	$t_{156}=0.00, p=.997$
	CBT+CM	-5.4	(-10.0, -0.8)	$t_{154}=2.34, p=.021$
6-month follow-up	CBT vs. CBT+CM	-5.4	(-12.7, +1.9)	$t_{155}=1.47, p=.144$
	CBT	-1.8	(-7.7, +4.0)	$t_{168}=0.62, p=.538$
	CBT+CM	-3.9	(-8.6, +0.7)	$t_{160}=1.67, p=.097$
	CBT vs. CBT+CM	-2.1	(-9.6, +5.4)	$t_{165}=0.56, p=.579$

Note. CBT = cognitive-behavioral treatment; CM = contingency management; AUC = Area Under the Curve.

^a = Actual AUC values have been multiplied by 100.

Table 5

Analysis of Variance of Discounting Change Measures

Accounting for CM and Smoking-status

	AUC			
	End-of-treatment		6-month follow-up	
	$F_{(1,110)}$	p	$F_{(1,101)}$	p
CM	0.17	.683	0.22	.637
Smoke	1.41	.238	0.03	.865
CM×Smoke	0.04	.846	0.02	.895

Note. CM = contingency management; Smoke = smoking or abstinent at each indicated assessment period; AUC = Area Under the Curve.

Table 6

Estimated Change from Intake Levels Among Smokers and Abstainers for Each Treatment Condition and Assessment Period

	AUC			
	End-of-treatment		6-month follow-up	
	CBT	CM+CBT	CBT	CM+CBT
Smokers	+3.7	+2.4	-1.0	-3.3
Abstainers	-2.2	-5.9	-2.1	-3.4
Difference	+6.0	+8.3	+1.1	+0.1
and 95% CI	(-6.4, +18.3)	(-12.1, +28.8)	(-10.8, +13.1)	(-9.0, +9.3)

Note. CBT = cognitive-behavioral treatment; CM = contingency

management; AUC = Area Under the Curve; actual AUC values have been multiplied by 100.

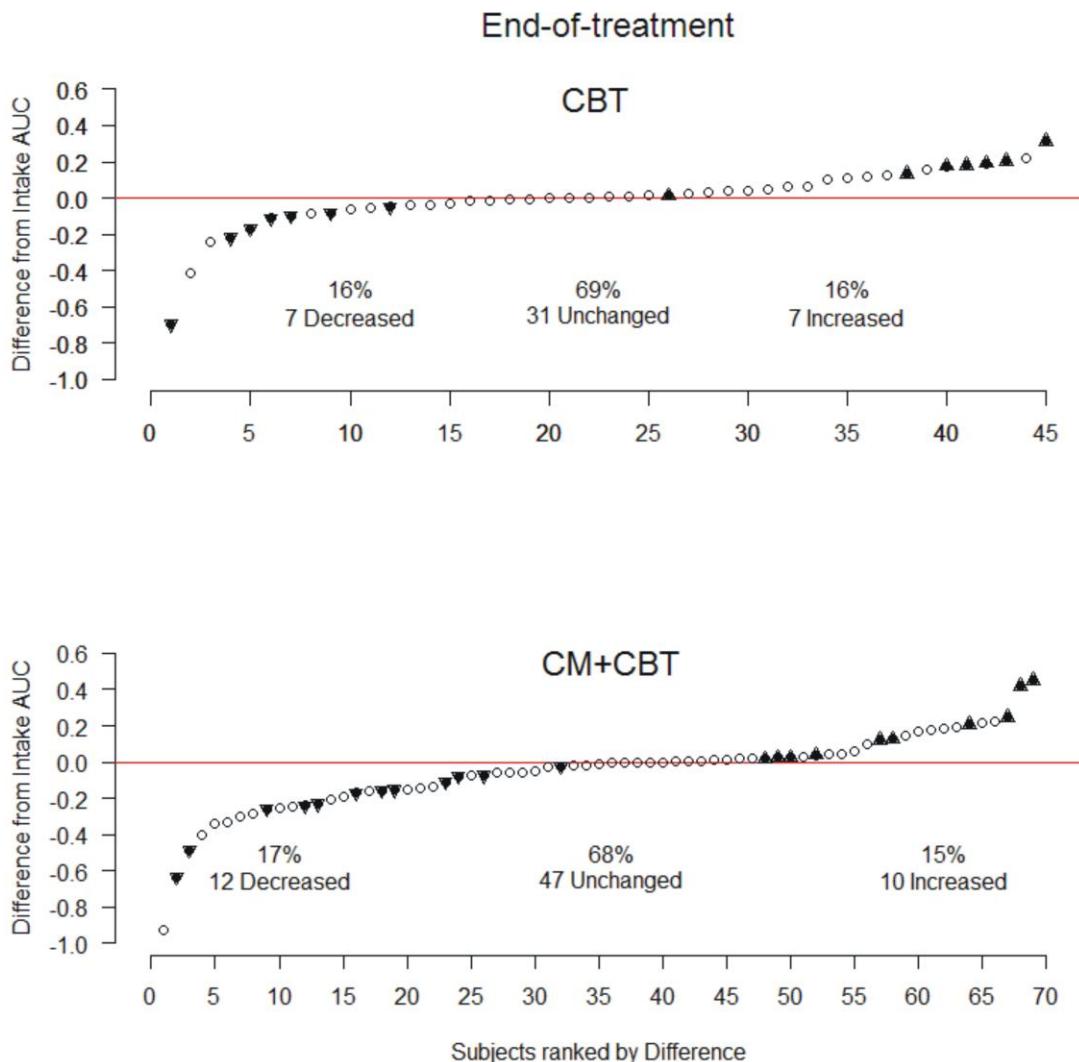


Figure 1. Within-individual change from intake AUC at end-of treatment among the CBT and CM + CBT groups. Circles represent no statistical change; triangles statistical increase; inverted triangles statistical decrease.

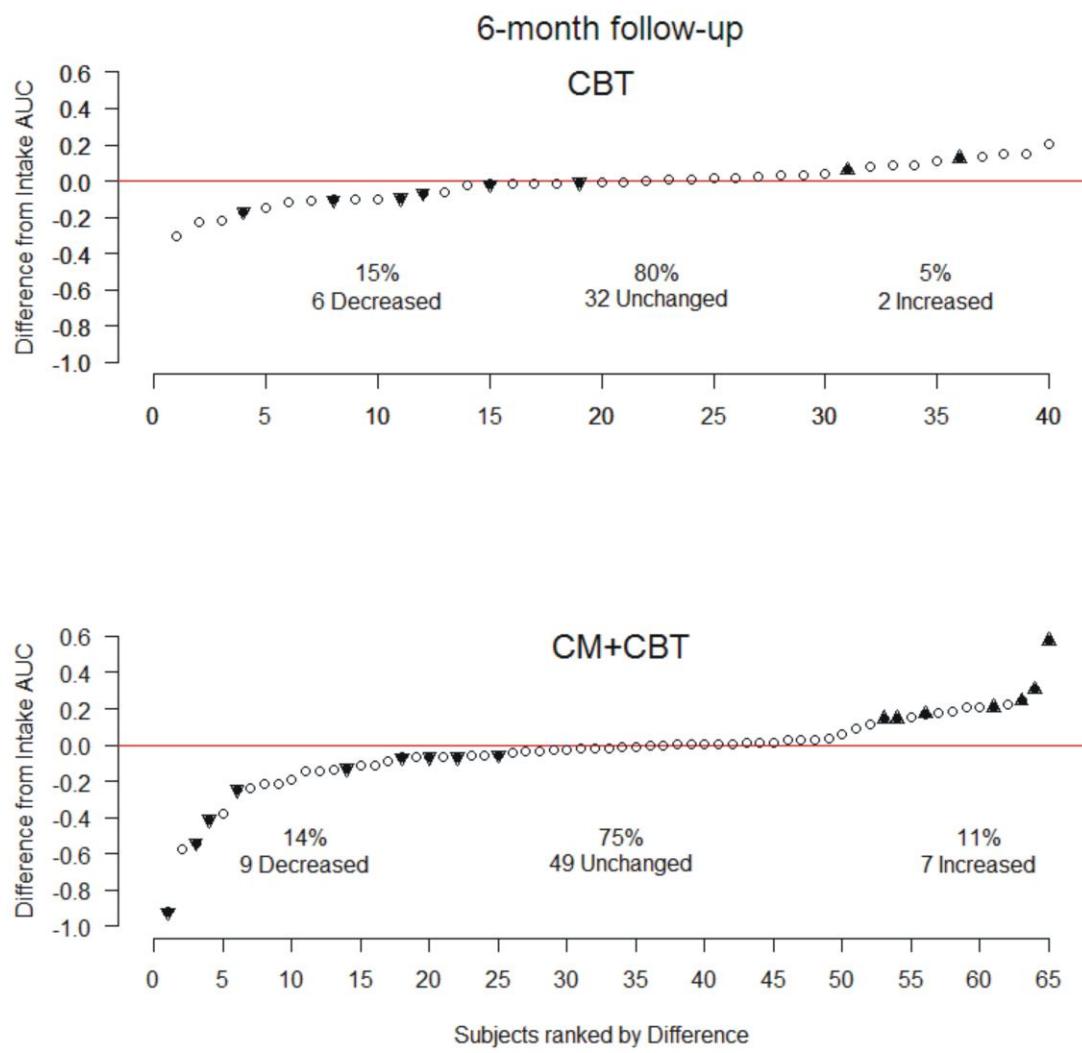


Figure 2. Within-individual change from intake AUC at 6-month follow-up among the CBT and CM + CBT groups. Circles represent no statistical change; triangles statistical increase; inverted triangles statistical decrease.

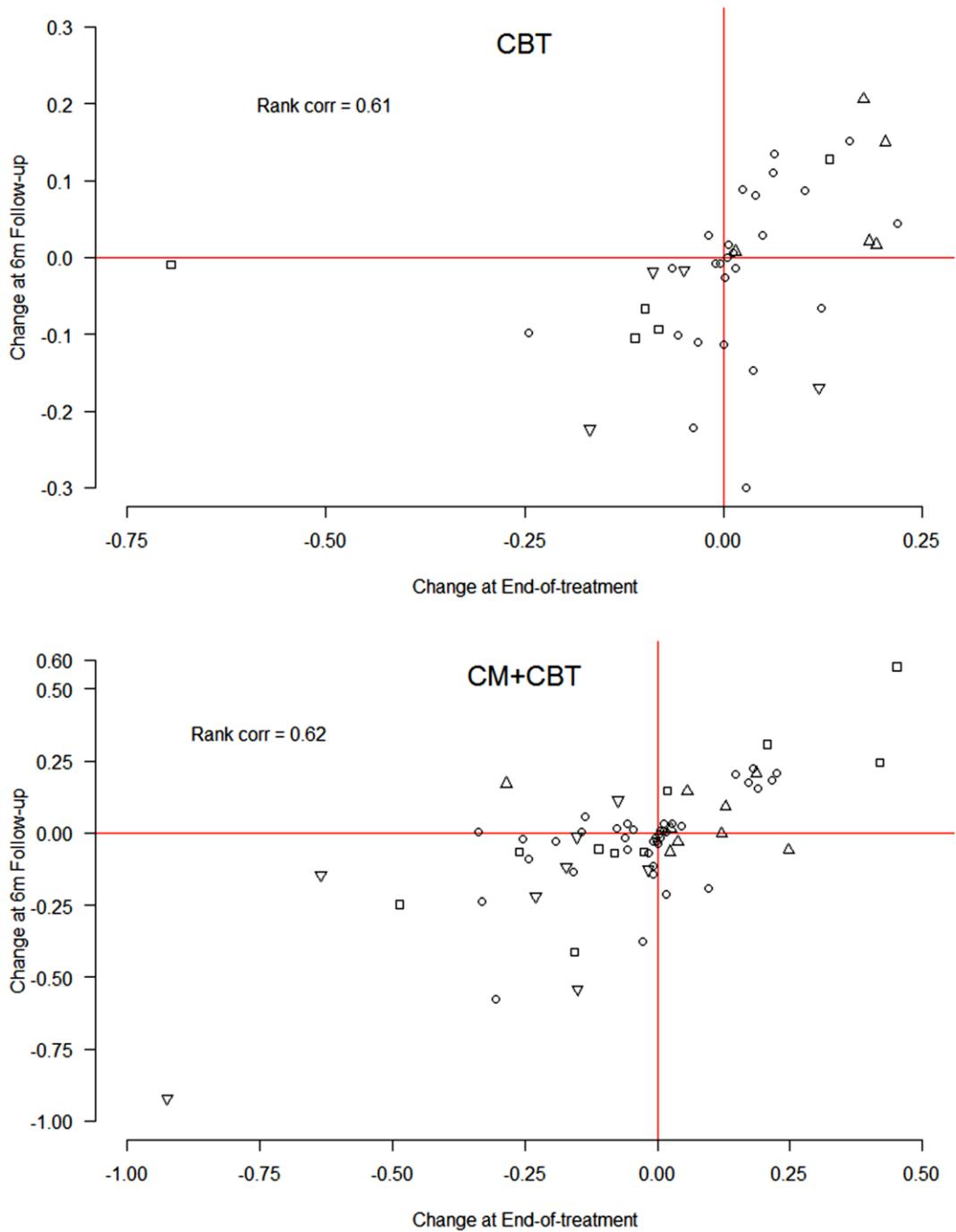


Figure 3. Within-individual change from intake AUC at end-of-treatment plotted by that at 6-month follow-up for CBT and CM + CBT. Circles represent no statistical change at either time point; squares statistical change at both time points; triangles statistical increase; inverted triangles statistical decrease.

5. DISCUSIÓN Y CONCLUSIONES

El objetivo general de la presente Tesis Doctoral fue la evaluación de la impulsividad a través de la aplicación de la tarea de descuento por demora en sujetos dependientes de sustancias. Dado que la modalidad de presentación de la Tesis Doctoral es el compendio de publicaciones, a continuación se recoge un resumen de las distintas secciones de la discusión correspondientes a cada uno de los cuatro objetivos específicos que conforman la Tesis Doctoral.

5.1. Evaluación de las tasas de descuento por demora en relación a la dependencia de cocaína y nicotina

El primer objetivo específico de la Tesis Doctoral fue la comparación de las tasas de descuento por demora de tres grupos de sujetos drogodependientes (sujetos dependientes de cocaína y nicotina, dependientes sólo de cocaína, dependientes sólo de nicotina) y un grupo control no dependiente de ninguna sustancia. A pesar del considerable número de estudios previos que mostraron que los sujetos dependientes tanto de sustancias legales como ilegales descuentan más por demora que los controles no dependientes (Baker, et al., 2003; Coffey, et al., 2003; Heil, et al., 2006; Kirby y Petry, 2004), la relación entre el descuento por demora y el policonsumo de sustancias no era clara. Los resultados de este estudio mostraron que los sujetos de los tres grupos dependientes de sustancias (grupo dependiente de cocaína y nicotina, grupo dependiente de cocaína sólo y grupo dependientes de nicotina sólo) tuvieron un mayor descuento que el grupo control. En segundo lugar, los dos grupos dependientes de la cocaína (ya sea el grupo dependiente de cocaína y nicotina o el grupo dependiente de cocaína sólo) presentaron un descuento mayor que el grupo de sujetos dependientes de nicotina sólo. Estos datos contrastan con el estudio de Businelle, et al. (2010) que encontró que los sujetos dependientes de nicotina y los de sujetos dependientes de diversas sustancias presentaban tasas de descuento por demora similares. Sin embargo, el estudio de Businelle, et al. (2010) presentaba la importante limitación relativa a la heterogeneidad de los sujetos policonsumidores en términos del tipo y el número de trastornos por consumo de sustancias presentado. Este resultado señala que el tipo de sustancia adictiva afecta a las diferencias en las tasas de descuento por demora. Por último, los sujetos dependientes de la cocaína y la nicotina, y los sujetos dependientes de la cocaína sólo no difirieron en sus tasas de descuento por demora. Es decir, el número de sustancias de las que los sujetos dependen no parece ser un factor determinante a la hora

de generar diferencias entre grupos en las tasas de descuento por demora. La revisión de Lundqvist (2005) concluye que los sujetos dependientes de cocaína muestran una mayor disminución de la capacidad atencional y de aprendizaje, así como mayores déficits de memoria de trabajo y flexibilidad cognitiva en comparación con otras drogas. En este sentido, las altas tasas de descuento por demora han demostrado estar asociadas con sesgos atencionales y con una menor capacidad de la memoria de trabajo (Field, Christiansen, Cole, y Goudie, 2007; Shamosh et al., 2008), lo que puede contribuir a explicar la influencia sustancial que la cocaína ejerce sobre las tasas de descuento por demora en comparación con la nicotina.

En resumen, el presente estudio señala que es el tipo y no tanto el número de sustancias el factor más determinante en las tasas de descuento por demora.

5.2. Evaluación de las tasas de descuento por demora en fumadores que reciben un tratamiento para dejar de fumar: resultados al final de tratamiento y al año de seguimiento

El segundo objetivo específico de la Tesis Doctoral fue analizar longitudinalmente si se producían cambios en las tasas de descuento por demora en el post-tratamiento y a los doce meses de seguimiento en función de la abstinencia o consumo de tabaco en fumadores que habían recibido un tratamiento para dejar de fumar. La naturaleza transversal de la mayor parte de estudios previos sobre descuento por demora impedía conocer si las altas tasas de descuento por demora existentes en la población drogodependiente eran un factor de riesgo para el desarrollo de trastornos por consumo de sustancias o más bien consecuencia de los mismos. Además, los resultados de los escasos estudios longitudinales llevados a cabo en población fumadora eran mixtos: un estudio mostró que las tasas de descuento por demora disminuían en fumadores que recibían un tratamiento de cinco días de duración para reducir el consumo de tabaco (Yi, et al., 2008), y dos estudios concluyeron que el descuento por demora se mantenía estable tras varias semanas (Yoon, Higgins, Bradstreet, Badger, y Thomas, 2009) o un año de abstinencia del tabaco (Yoon, et al., 2007). El presente estudio encontró que el descuento se mantuvo estable tanto en los participantes que se encontraban abstinentes como en aquellos que continuaban fumando tras finalizar un tratamiento para dejar de fumar. Sin embargo, el descuento por demora se redujo en los abstinentes al año de seguimiento, mientras que continuó manteniéndose estable entre

los fumadores. Estos resultados sugieren que se requiere un periodo largo de abstinencia del tabaco para detectar cambios en el descuento por demora asociados al abandono del consumo, y apoyan la idea de que el descuento por demora constituye una variable estado susceptible de ser modificada (Field, et al., 2006; Giordano, et al., 2002; Reynolds, 2004; Yi, et al., 2008). La abstinencia prolongada del tabaco puede producir ciertos cambios tales como la adopción de un estilo de vida más saludable (Melanko y Larkin, 2013), una mayor actividad de los circuitos neuronales del sistema ejecutivo (Bickel et al., 2007), y una disminución de los sesgos atencionales hacia las claves relacionadas con el consumo de tabaco (Munafo, Mogg, Roberts, Bradley, y Murphy, 2003), modificaciones que a su vez pueden redundar en una reducción del descuento por demora.

La principal implicación clínica derivada de los resultados de este estudio tiene que ver con la importancia que las intervenciones para dejar de fumar han de otorgar a las consecuencias a corto plazo derivadas de fumar, en vez de focalizarse en aquellas a largo plazo. En este sentido, el Manejo de Contingencias ha demostrado ser un tratamiento eficaz para el tabaquismo (Alessi, Badger, y Higgins, 2004; Dallery, Glenn, y Raiff, 2007), proporcionando reforzadores inmediatos de forma contingente a la abstinencia del tabaco. No obstante, dadas las recaídas que se producen una vez finalizado el MC (Ledgerwood, 2008), son necesarios estudios adicionales que exploren nuevas vías de promover la abstinencia más allá de este tratamiento.

En síntesis, los resultados encontrados indican que las altas tasas de descuento por demora observadas en la población fumadora pueden reducirse mediante el logro de la abstinencia a largo plazo.

5.3. Análisis de los efectos principales y de interacción de la sintomatología depresiva y los cambios en el estatus de consumo de tabaco en las tasas de descuento por demora en fumadores que reciben un tratamiento para dejar de fumar

El tercer objetivo específico de la Tesis Doctoral fue evaluar los efectos principales e interactivos de los síntomas depresivos y los cambios en el estatus de consumo de tabaco sobre el descuento por demora. Los resultados mostraron que los participantes con y sin sintomatología depresiva no difirieron en sus tasas de descuento por demora ni al inicio del tratamiento ni a los seis meses de seguimiento. Este

resultado apoya los hallazgos de estudios previos (Dennhardt y Murphy, 2011; Dombrovski, et al., 2011; V. M. Gonzalez, Reynolds, y Skewes, 2011), y sugiere que es posible que solamente aquellos individuos que cumplen criterios diagnósticos de depresión mayor tengan altas tasas de descuento por demora, mientras que la sintomatología depresiva no parece tener efecto sobre el descuento por demora (Takahashi et al., 2008). Por otro lado, las tasas de descuento fueron más bajas entre los participantes que estaban abstinentes a los 6 meses de seguimiento en comparación con aquellos que continuaban fumando. Si se tienen en cuenta los resultados tanto del presente estudio como del cuarto estudio, se puede concluir que es posible que la abstinencia a medio plazo tenga un cierto efecto sobre las tasas de descuento por demora, aunque desde luego no tan evidente como el efecto de la abstinencia a largo plazo. Por último, la sintomatología depresiva moderó la relación entre el descuento por demora y el estatus de consumo de tabaco a los seis meses de seguimiento, de tal forma que el efecto de la abstinencia sobre la reducción del descuento por demora se encontraba acentuado en los participantes abstinentes con síntomas depresivos. Varios factores pueden explicar este efecto interactivo de la sintomatología depresiva y el estatus de consumo de tabaco sobre el descuento por demora. Entre ellos, una alta aversión al riesgo observada entre la población abstinente del tabaco (Goto, Takahashi, Nishimura, y Ida, 2009) y con sintomatología depresiva (Smoski et al., 2008) puede contribuir a unas tasas de descuento por demora inferiores. Así mismo, la adopción de un estilo de vida más saludable debido a la abstinencia del tabaco ha demostrado estar exacerbada en los individuos con síntomas depresivos (C. A. Green y Pope, 2000), lo que permite observar las bajas tasas de descuento por demora en los individuos que abstinentes a los seis meses de seguimiento y que presentan sintomatología depresiva.

Una importante implicación clínica derivada de este estudio guarda relación con las altas de descuento por demora encontradas entre los participantes con sintomatología depresiva que continuaban fumando a los seis meses de seguimiento. Dado que estos individuos parecen estar especialmente influenciados por las consecuencias inmediatas derivadas de la conducta de fumar en detrimento de las demoradas, aquellos tratamientos que incorporen reforzadores inmediatos para la abstinencia del tabaco, tales como el Manejo de Contingencias, pueden ser especialmente útiles entre los fumadores con sintomatología depresiva. A este respecto, varios estudios previos señalan la eficacia del MC en el tratamiento de individuos con un trastorno por

consumo de sustancias que también presentan un diagnóstico de depresión (Drebing et al., 2005; G. Gonzalez, Feingold, Oliveto, Gonsai, y Kosten, 2003).

En síntesis, los hallazgos del presente estudio muestran que la reducción de la toma de decisiones impulsiva asociada a la abstinencia del tabaco se encuentra acentuada en los individuos que presentan sintomatología depresiva.

5.4. Efectos del Manejo de Contingencias (MC) sobre el descuento por demora en fumadores que reciben un tratamiento para dejar de fumar

El cuarto objetivo específico de la Tesis Doctoral fue evaluar el efecto diferencial del MC añadido a un Tratamiento Cognitivo Conductual (TCC) para dejar de fumar, sobre el descuento por demora al final del tratamiento y a los seis meses de seguimiento. A pesar de la evidencia creciente que sugería que el descuento por demora se reducía tras la aplicación de intervenciones efectivas, los resultados relativos al efecto del MC en el descuento por demora eran mixtos; mientras que dos estudios indicaban que el MC reducía el descuento por demora en fumadores (Yi, et al., 2008) y en individuos dependientes de opiáceos (Landes, et al., 2012), otro estudio señalaba que el descuento por demora se mantenía estable en sujetos dependientes de marihuana que también recibían MC (Peters, et al., 2013). Los análisis entre-grupos mostraron que no había diferencias en el descuento por demora entre el grupo de MC + TCC y el grupo de TCC ni al final del tratamiento, ni a los seis meses de seguimiento. No obstante, los análisis intra-grupos revelaron una cierta reducción del descuento por demora a lo largo del tiempo en el grupo de MC + TCC, mientras que éste se mantuvo estable en el grupo de TCC. Por otro lado, los análisis individuales mostraron que el porcentaje de sujetos cuyo descuento por demora aumentaba, se mantenía o disminuía a lo largo del tiempo era igual en ambas grupos de tratamiento. Por último, la abstinencia del tabaco no tuvo efectos en el descuento por demora ni al final del tratamiento ni a los seis meses de seguimiento. No obstante, se observó una tendencia por la cual los abstinentes presentaban tasas de descuento por demora más bajas que los fumadores. En general, la mayor parte de los resultados indican que ni el MC+ TCC ni el TCC modifican el descuento por demora. Este hallazgo sugiere que es posible que ciertas intervenciones focalizadas en las funciones ejecutivas (ver Bickel, Yi, et al., 2011) o en el empleo de potenciadores cognitivos a través de tratamientos psicofarmacológicos (Bickel, Jarmolowicz, Mueller, Koffarnus, y Gatchalian, 2012) pueden ser más eficaces para

reducir el descuento por demora. No obstante, el hecho de que el análisis intra-grupos evidenciara una cierta reducción del descuento por demora a lo largo del tiempo en el grupo de MC + TCC puede deberse a un descenso más pronunciado a nivel individual en aquellos participantes que recibieron MC y que mostraron una reducción de su descuento por demora con respecto al inicio del tratamiento, en comparación con los participantes cuyo descuento por demora se redujo pero no recibieron MC. Por otro lado, el hecho de que la abstinencia tanto a corto plazo (al final del tratamiento) como a medio plazo (a los seis meses de seguimiento) no tuviera efecto en las tasas de descuento por demora apoya los resultados de estudios previos (Dallery y Raiff, 2007; Yoon, et al., 2009; Yoon, et al., 2007) y sugiere que son necesarios períodos de abstinencia más largos (de al menos un año como se observa en el segundo estudio de la presente Tesis Doctoral) para detectar cambios en el descuento por demora.

En resumen, los resultados sugieren que el MC no parece estar asociado de forma robusta con cambios en el descuento por demora.

5.5. Limitaciones

En este apartado se resumen las principales limitaciones de los estudios de la Tesis Doctoral. En primer lugar se mencionan las limitaciones de carácter general que atañen a todos los estudios, y después se comentan las limitaciones específicas de los mismos.

Por lo que respecta a las limitaciones generales, se debe señalar que se han empleado reforzadores monetarios hipotéticos en vez de reales en el procedimiento de descuento por demora. No obstante, un número considerable de estudios previos encuentran resultados similares al comparar las tasas de descuento por demora empleando reforzadores monetarios reales frente a hipotéticos (Dixon, Lik, Green, y Myerson, 2013; Johnson y Bickel, 2002; Lagorio y Madden, 2005; Madden, Begotka, Raiff, y Kastern, 2003; Madden, et al., 2004).

En segundo lugar, existen ciertas variables individuales que han demostrado influir en el descuento por demora y que no se han controlado en los estudios, tales como la inteligencia (Shamosh et al., 2008), la historia familiar de dependencia de sustancias (Petry, Kirby, y Kranzler, 2002) o el género (Reynolds, Ortengren, et al., 2006).

El tamaño muestral relativamente reducido es una limitación importante que dificulta la generalización de los resultados y la posibilidad de encontrar diferencias estadísticamente significativas en algunas comparaciones. Este problema afecta

especialmente al primer y tercer estudio de la Tesis Doctoral. En relación al primer estudio, el reducido tamaño muestral en el grupo de sujetos dependientes de cocaína sólo ($n = 20$) puede haber limitado la capacidad para encontrar diferencias significativas en el descuento por demora entre este grupo y el grupo de sujetos dependientes de la cocaína y nicotina. Relativo al tercer estudio, el número de participantes con sintomatología depresiva también fue reducido ($n = 20$). No obstante, esto no supuso un impedimento para detectar un efecto interactivo estadísticamente significativo de la sintomatología depresiva y el estatus de consumo de tabaco sobre las tasas de descuento por demora.

Por último, los fumadores incluidos en los estudios presentaban una dependencia moderada del tabaco (con un máximo de 23.66 cigarrillos fumados al día y 5.5 en el test de Fagerström de dependencia a la nicotina), lo que puede limitar la posibilidad de generalizar los resultados encontrados a fumadores con diferentes niveles de dependencia.

Las limitaciones específicas de los estudios son las siguientes:

El primer estudio se encuentra limitado por el hecho de que la muestra estuvo compuesta exclusivamente por hombres, lo que puede reducir la capacidad de generalización de los resultados encontrados. El tiempo transcurrido desde el último consumo de cocaína (de 0 a 90 días) no se controló en el primer estudio debido al reducido tamaño muestral de los grupos dependiente de cocaína y nicotina y dependiente de cocaína sólo.

En el segundo estudio no se incluyó un grupo de nunca fumadores que hubiera permitido analizar si las tasas de descuento por demora de los participantes abstinentes al año de seguimiento se reducen de hecho a niveles de sujetos que nunca han fumado. Se empleó una medida de punto de prevalencia de siete días para definir la abstinencia del tabaco en los diferentes seguimientos, en vez de utilizar una medida más precisa de abstinencia continuada. Esta limitación afecta a los estudios segundo, tercero y cuarto.

En el tercer estudio no se empleó una entrevista clínica estructurada (como el SCID-II) para evaluar depresión mayor. No obstante, se utilizó el BDI-II, un instrumento fiable y válido para evaluar sintomatología depresiva y que presenta buenas propiedades psicométricas (Hides, Samet, y Lubman, 2010).

5.6. Conclusiones

Las conclusiones generales que se pueden extraer de los estudios llevados a cabo en la presente Tesis Doctoral son las siguientes:

- 1) El tipo de sustancia adictiva parece ser determinante en las tasas de descuento por demora. En sentido contrario, el número de sustancias de las que los sujetos dependen no constituye una variable que afecte al descuento por demora.
- 2) Un periodo corto de abstinencia del tabaco no tiene efectos sobre el descuento por demora. Sin embargo, las tasas de descuento por demora de los fumadores pueden reducirse mediante el logro de la abstinencia a largo plazo.
- 3) Los individuos abstinentes y los que continuaban fumando al año de seguimiento no difirieron en sus tasas de descuento por demora en la línea base. Sin embargo, la abstinencia del tabaco a largo plazo redujo las tasas de descuento por demora.
- 4) Los fumadores con y sin síntomas de depresión no difirieron en el descuento por demora ni en la línea base ni a los 6 meses de seguimiento.
- 5) La sintomatología depresiva influye en la relación entre el descuento por demora y el estatus de consumo de tabaco. En concreto, la reducción de las tasas de descuento por demora asociada a la abstinencia del tabaco fue más acentuada en los participantes abstinentes con síntomas de depresión.
- 6) Una intervención de MC no está asociada de forma robusta con modificaciones en las tasas de descuento por demora entre la población fumadora.

5.7. Conclusions (bis)

The general conclusions that can be derived from the studies conducted in the present Doctoral Thesis are the following:

- 1) The type of substance which individuals depend on seems to be decisive in delay discounting. Contrary, the number of substances which individuals depend on does not seem to affect delay discounting.
- 2) Short term abstinence has no effect on delay discounting. However, smokers' delay discounting rates can be reduced with the achievement of long term abstinence.
- 3) Abstinent individuals and those who continued smoking at one year follow-up did not differ in their baseline delay discounting rates. However, long term abstinence reduced delay discounting.

- 4) Smokers with and without depressive symptoms did not differ in their delay discounting either at baseline or at 6-month follow-up.
- 5) Depressive symptomatology influenced the relationship between delay discounting and smoking status. In particular, delay discounting reduction associated with smoking abstinence was enhanced among abstinent participants with depressive symptoms.
- 6) A CM intervention was not robustly associated with delay discounting changes among the smoking population.

5.8. Líneas futuras de investigación

En este último apartado se recogen algunas orientaciones para la puesta en marcha de nuevos estudios sobre la evaluación del descuento por demora en sujetos dependientes de sustancias.

- 1) Mientras que los estudios previos de tipo transversal mostraban que los fumadores de tabaco presentaban tasas de descuento por demora superiores a los ex fumadores (Bickel, et al., 1999; Sweitzer, et al., 2008), los resultados transversales relativos al efecto de la abstinencia de otras drogas como la heroína (Bretteville-Jensen, 1999), la cocaína (Heil, et al., 2006) o el alcohol (Petry, 2001) sobre el descuento por demora son inconsistentes. El segundo estudio longitudinal de la presente Tesis Doctoral permite concluir que las altas tasas de descuento por demora observadas en la población fumadora son una consecuencia de la dependencia de la nicotina y pueden reducirse con la abstinencia prolongada. Dada la inconsistencia de los estudios transversales llevados a cabo con otras sustancias aparte de la nicotina, son necesarios nuevos estudios que analicen si la abstinencia del consumo de otras drogas produce reducciones en las tasas de descuento por demora, al igual que ocurre con la población fumadora.
- 2) Los estudios de la Tesis Doctoral han sido realizados con fumadores con dependencia moderada. Dado que la gravedad de la dependencia a la nicotina ha demostrado ser una variable que influye en las tasas de descuento por demora (Johnson, et al., 2007; Rezvanfard, et al., 2010), son necesarios nuevos estudios que incluyan a fumadores con diferentes niveles de dependencia.
- 3) En los estudios de la presente Tesis Doctoral se han excluido a los individuos con algún trastorno psicopatológico grave. Diferentes trastornos

psicopatológicos tales como la esquizofrenia (Heerey, Robinson, McMahon, y Gold, 2007; Weller, et al., 2014), el trastorno bipolar (Ahn et al., 2011) o el trastorno por estrés postraumático (Engelmann, Maciuba, Vaughan, Paulus, y Dunlop, 2013) han demostrado afectar a las tasas de descuento por demora, por lo que sería conveniente la realización de nuevas investigaciones con población drogodependiente que presenta trastornos psicopatológicos graves.

- 4) Aunque la investigación previa ha encontrado altas correlaciones entre las medidas de punto de prevalencia y de abstinencia continuada (Hughes, Carpenter, y Naud, 2010; Velicer y Prochaska, 2004), Hughes et al. (2003) recomiendan el empleo ambas medidas en los ensayos clínicos que realizan intervenciones para dejar de fumar, usando la abstinencia continuada como variable principal y el punto de prevalencia como variable secundaria. En los estudios de esta Tesis Doctoral solamente se empleó una medida de punto de prevalencia de siete días para definir la abstinencia del tabaco en los diferentes seguimientos. Futuros estudios han de analizar si se obtienen los mismos resultados cuando se emplea la abstinencia continuada o se usan ambas medidas.
- 5) En el tercer estudio de la Tesis Doctoral solamente se empleó el BDI-II para evaluar sintomatología depresiva. Dado que la investigación previa ha demostrado que el trastorno por depresión mayor afecta a las tasas de descuento por demora (Pulcu et al., 2014; Takahashi, et al., 2008), sería interesante que futuros estudios analizaran el efecto de la abstinencia del tabaco sobre las tasas de descuento por demora en población fumadora que presenta depresión mayor.
- 6) Por último, serían necesarios nuevos estudios que analizasen qué tratamientos o combinaciones de sus componentes son más eficaces para reducir la toma de decisiones impulsiva en la población drogodependiente. Específicamente, la investigación previa señala que ciertas intervenciones focalizadas en las funciones ejecutivas (ver Bickel, Yi, et al., 2011) o en el empleo de potenciadores cognitivos a través de tratamientos psicofarmacológicos (Bickel, Jarmolowicz, Mueller, Koffarnus, y Gatchalian, 2012) pueden ser más eficaces para reducir el descuento por demora.

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