

Neuropsychological impairments associated with the relation between cocaine abuse and violence: neurological facilitation mechanisms

Déficits neuropsicológicos asociados a la relación entre abuso de cocaína y violencia: mecanismos neuronales facilitadores

ROMERO-MARTÍNEZ, A.; MOYA-ALBIOL, L.

Department of Psychobiology, Universitat de València, Valencia

Abstract

Introduction. Cocaine abuse, as well as prenatal exposure to cocaine, could be key factors in the expression of violent behaviour. Neuropsychological impairments, sex differences and the concurrent abuse of cocaine and alcohol have been suggested as facilitation mechanisms. **Aims.** To review and recapitulate the results obtained on the relationship between neuropsychological deficits due to cocaine abuse and/or prenatal exposure and the expression of violence. Furthermore, we analyze the roles of sex, concurrent alcohol abuse and possible brain damage as risk markers in this relationship. **Development.** The scientific literature was reviewed using Google Scholar, PsycINFO, PubMed, Medline and ISI Web of Knowledge. **Conclusions.** Cocaine facilitates the expression of violence due to neuropsychological deficits in emotional decoding, abstract reasoning and inhibitory control, as well as in mnemonic and verbal skills, and such impairments might also explain problems in decision-making. Both the deficits and the expression of violence appear to be more pronounced in men than in women. However, despite the fact that the combination of cocaine and alcohol use may increase the risk of violent reactions, the deficits would not be greater than those resulting from the separate use of each substance. The impairments might be caused by functional abnormalities of certain regions of the frontal (especially the prefrontal) and parietal lobes and some subcortical structures, such as the amygdala. All of this would provide a basis for the development of intervention strategies focusing on these cognitive domains.

Keywords: alcohol, cocaine, gender, neuropsychology, violence.

Resumen

Introducción. El abuso de la cocaína, así como la exposición prenatal a la misma parece ser un factor relevante en el desencadenamiento de comportamientos violentos. Los déficits neuropsicológicos, así como el género y la combinación con el alcohol, serían los posibles mecanismos facilitadores. **Objetivo.** Revisar y recapitular los resultados obtenidos sobre los déficits neuropsicológicos debidos al abuso o a la exposición prenatal a la cocaína y relacionarlos con la expresión de la violencia. Además, se enfatiza el papel del género y el abuso del alcohol junto a la cocaína, así como la posible existencia de daño orgánico cerebral como mecanismos facilitadores. **Desarrollo.** Se ha revisado la bibliografía científica usando los buscadores Google Scholar, PsycINFO, PubMed, Medline e ISI Web of Knowledge. **Conclusiones.** La cocaína facilitaría la expresión de la violencia debido a los déficits en la decodificación emocional, la capacidad de abstracción e inhibición, así como en las habilidades verbales y mnémicas. Esto explicaría, además, los problemas en la toma de decisiones. Los déficits y la expresión de la violencia parecen ser más evidentes en los hombres. Sin embargo, a pesar de que la combinación de la cocaína con el alcohol incrementaría el riesgo de reaccionar de forma violenta, los déficits no serían mayores que el consumo de cada una de ellas por separado. Estos déficits podrían ser producto de un funcionamiento anormal de algunas áreas del lóbulo frontal (especialmente el prefrontal) y el parietal, así como estructuras subcorticales como la amígdala. Todo ello permitiría planificar estrategias de intervención cuyos objetivos serían estos dominios cognitivos.

Palabras clave: alcohol, cocaína, género, neuropsicología, violencia.

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Address for correspondence:

Dr. Luis Moya-Albiol. Department de Psicobiologia, Universitat de València. Avenida Blasco Ibañez, 21, 46010, Valencia (Spain).
Email: Luis.Moya@uv.es.

Substance addiction is known to be a key factor in the triggering of antisocial and/or violent behaviour. However, addictions are not always linked to violence, and addictions can be found without the manifestation of violence, since there is no causal relation between them (Gómez et al., 2008; Romero-Martínez, & Moya-Albiol, 2013). Indeed, violent behaviour tends to precede drug use (Collins & Messerschmidt, 1993; Farrington, 1994).

Several studies have reported a positive relationship between cocaine use and violent behaviour (Brody, Slovis, & Wrenn, 1990; Moore et al., 2008; Kraanen, Vedel, Scholling, & Emmelkamp, 2014; Pennings, Leccese, & Wolff, 2002), as well highlighting the seriousness of such behaviour (Chermack & Blow, 2002). Violent responses can be facilitated either directly, as a result of acute cocaine use (Licata, Taylor, Berman, & Cranston, 1993) or indirectly, due to cognitive deficits caused by high prenatal exposure to this substance (Bendersky & Lewis, 1998; 2001; Mayes, Bornstein, Chawarska, Haynes, & Granger, 1996), or by its chronic use in adulthood (Volkow et al., 1997).

Both sex and poly-drug use (including cocaine) are considered as triggers for violence (Delaney-Black et al., 2004). The relationship between cocaine use and violence is more evident in men (Chermack et al., 2010); also, cocaine and alcohol tend to be consumed at the same time (Alcázar-Córcoles & Bezos-Saldaña, 2011; Chermack et al., 2008; Chérrez-Bermejo & Alás-Brun, 2014), with the result that the euphoria-inducing effects of cocaine use increase in duration and intensity, increasing in turn the risk of violent reactions (Heinz, Beck, Meyer-Lindenberg, Sterzer, & Heinz, 2011; Lizasoain, Moro, & Lorenzo, 2001; McCance-Katz, Kosten, & Jatlow, 1998).

The absence of a well-defined and systematized theoretical framework regarding the mediating factors between cocaine use/abuse/dependence and the triggering of violence, and more specifically regarding the neuropsychological deficits that can facilitate violent reactions, hinders a comprehensive understanding of the mechanisms involved. In the light of the above, and with the aim of providing a synthesis of the scientific literature focusing on the analysis of mediating variables between the exposure to or consumption of cocaine and the facilitation of aggressive behaviour, we shall first describe the main findings on the neuropsychological domains altered in people that abuse or are dependent on cocaine and those that were prenatally exposed to this substance. Next, we shall discuss the most relevant findings on the role of the main contributory variables, such as sex and alcohol and cocaine abuse/dependence. Finally, and taking into account previous data on the altered functioning of several brain structures underlying these deficits, we shall analyze the possible existence of organic damage or hypo-functionality in the central nervous system (CNS).

Parameters of the literature search

We have Google Scholar, PsycINFO, PubMed, Medline and ISI Web of Knowledge to review the scientific literature on the relationship between neuropsychological deficits and the expression of aggressive behaviour in cocaine users. The initial search encompassed the following terms: *aggressive behaviour*, *cocaine*, *biological correlates*, *cognitive deficits*, *empathy*, *emotion recognition*, *executive functions*, *intelligence*, *neuropsychology* and *violence*. Articles dealing with biological variables only, with no direct or indirect mention of the expression of violence, were not considered in our review.

Neuropsychological domains

Empathy

Cocaine-dependent subjects have empathy-related deficits, specifically in perspective-taking, emotional decoding and emotional empathy (Kemmis, Hall, Kingston, & Morgan, 2007; Preller et al., 2014; Roselli & Ardila, 1996; Verdejo-García, Rivas-Perez, Vilar-López, & Pérez-García, 2007). In addition, they present high levels of alexithymia, that is, deficits in the ability to identify and verbalize their own emotions (Keller, Carroll, Nick, & Rounsaville, 1995; Li & Sinha, 2006).

Emotional decoding or recognition processes (of both facial expressions and prosody) are essential for cognitive empathy and for inferring one's own and others' thoughts, intentions and feelings (Babcock, Green, & Webb, 2008; Kemmis, Hall, Kingston, & Morgan, 2007; Preller et al., 2014; Roselli & Ardila, 1996; Verdejo-García et al., 2007), and so determining subsequent behaviour (Calder & Young, 2005). In this regard, greater severity of cocaine use has been associated with poorer emotional recognition. More specifically, the greater the level of cocaine use and the higher the number of years of its consumption, the poorer the recognition of facial expressions of fear and anger (Fernández-Serrano, Lozano, Pérez-García, & Verdejo-García, 2010). Research results also show that young adults who use cocaine occasionally and/or for recreational purposes, together with users currently abstinent, presented particular deficits in the recognition of fear in the "eyes task", which assesses the extent to which people can "read the mind" from the other's facial expression (Kemmis et al., 2007). Furthermore, such deficits could be relatively stable, since they have been observed in users of several substances (cocaine among them) in abstinence periods ranging from 3 to 20 months (Foisy et al., 2007).

Studies in children prenatally exposed to cocaine are in the same line, as 3- to 6-year-olds that were prenatally exposed displayed less reactivity to the crying of other children and of their own mothers. They even showed less competence in cooperation tasks than non-prena-

tally exposed children (Jones, Field, Davalos, & Hart, 2004).

Deficits in empathic processes, as well as lack of remorse, have often been associated with antisocial behaviour (Moya-Albiol, Herrero, & Bernal, 2010; Preller et al., 2014). A possible explanation for this is that deficits in basic empathic processes, such as those of emotional decoding, are related to poor emotional regulation (Schipper & Petermann, 2013), which in turn affects decision-making. Moreover, cocaine users show poorer emotional and behavioural regulation during early abstinence (Fox, Axelrod, Paliwal, Sleeper, & Sinha, 2007). This might facilitate the expression of violence in these people, who fail to foresee and correctly interpret the consequences of their own actions (Blair, 2003).

Children prenatally exposed to cocaine have poor impulse control and emotion regulation and higher irritability than non-cocaine-exposed children (Bendersky & Lewis, 1998; 2001; Campbell, Bliven, Silveri, Snyder, & Spear, 2000; Fox, Calkins, Schmidt, Rubin, & Coplan, 1996; Mayes et al., 1996). Such poor behavioural regulation could increase the likelihood of their manifesting sustained disruptive behaviour, which would become more evident and serious as the socialization of the child/teenager runs its course over time (Allen, Bennett, Carmody, Wang, & Lewis, 2014; Rao et al., 2007).

Executive functioning

Chronic use of cocaine is related to poorer executive functioning, affecting capacity for inhibition, mental flexibility, planning ability, alternation of cognitive sets and decision-making (Colzato et al., 2009; Madoz-Gúrpide, Blasco-Fontecilla, & Baca-García, 2011; Morie, De Sanctis, & Foxe, 2014; Pike, Stoopsa, Fillmore, & Rush, 2013; Verdejo-García & Pérez-García, 2007; van der Plas et al., 2009; Woicik et al., 2009; 2011). This altered functioning has not only been found in adults: children and teenagers prenatally exposed to high levels of cocaine also present such deficits (Betancourt et al., 2011; Bridgett & Mayes, 2011; Grewen et al., 2014; Landi et al., 2012).

The executive functions are critical for to good social adaptation, so that deficits in these mental processes facilitate the expression of violence (Krämer, Kopyciok, Richter, Rodriguez-Fornells, & Münte, 2011; Raaijmakers et al., 2008). Whilst studies in adults that use cocaine and are violent have focused on the analysis of decision-making and mental flexibility, research in children and adolescents teenagers prenatally exposed to high levels of this substance have focused on inhibition processes.

One study revealed that cocaine users with higher levels of violence and antisocial traits committed fewer perseverative errors in the Wisconsin card sorting test than those who were less violent (Rosse, Miller, & Deutsch, 1993). The same study also suggested that a greater men-

tal flexibility (or fewer perseverations) in cocaine-abusing subjects with antisocial traits and high levels of violence could increase the chances of their committing an offence but avoiding any negative consequences of such behaviour (Rosse et al., 1993). However, the results are at odds with those for other subgroups of violent individuals (men serving sentences for domestic abuse and psychiatric population), which tend to show less mental flexibility (Miura, 2009; Romero-Martínez, Lila, Catalá-Miñana, Williams, & Moya-Albiol, 2013; Romero-Martínez, Lila, Sariñana-González, González-Bono, & Moya-Albiol, 2013; Romero-Martínez & Moya-Albiol, 2013). This could be explained by methodological errors in the mentioned study, by inadequate sample size or even by the instruments used in the assessment of antisocial traits.

A study that assessed decision-making through the Iowa Gambling Task revealed that abstinent cocaine users showed a behavioural pattern similar to those of violent subgroups, such as sex offenders, drug dealers and people arrested for drunk-driving. These people gave great importance to gains and rewards while disregarding losses and punishments (Yechiam et al., 2008). Thus, they would have deficits in reward processing, failing to consider all the relevant information and focusing only on that related to the greatest and most immediate rewards, and dismissing losses (Yechiam et al., 2008).

Children and teenagers exposed to cocaine during their mother's pregnancy often have a difficult temperament (Bendersky, Bennett, & Lewis, 2006; Moilanen, Shaw, & Fitzpatrick, 2010), which can lead to behavioural issues such as externalizing behaviour and/or risk or criminal behaviour (Allen et al., 2014; Bennett, Marini, Berzenski, Carmody, & Lewis, 2013; Delaney-Black et al., 2000; Min et al., 2014). Poor inhibitory control might explain this, since the lower the capacity for inhibitory control, the more serious the violence perpetrated (Bendersky et al., 2006; Carmody, Bennett, & Lewis, 2011; Holler & Kavanaugh, 2013; Pawliczek et al., 2013; Schafer & Fals-Stewart, 1997).

Memory

Several studies have revealed that adult cocaine users have deficits in memory, both immediate and delayed. They also have working memory deficits (Fox, Jackson, & Sinha, 2009; Spronk, van Wel, Ramaekers, & Verkes, 2013; Woicik et al., 2009), whose functions provide the foundation for other high-level cognitive processes, such as executive functioning (McCabe, Roediger, McDaniel, Balota, & Hambrick, 2010). These results have been replicated in children and teenagers that were prenatally exposed to high levels of cocaine (Bridgett & Mayes, 2011; Buckingham-Howes, Berger, Scaletti, & Black, 2013; Riggins et al., 2012). Moreover, such children show delayed development in the aforementioned abilities compared

to controls of the same age (Betancourt et al., 2011; Buckingham-Howes et al., 2013).

With regard to the relationship between cocaine use, memory deficits and violence, only one study has analyzed it in adults, reporting that in heterosexual couples with a history of poly-substance abuse (cocaine included), the greater the deficit in the delayed recollection of words in the *California Verbal Learning Test*, the poorer the recollection of episodes of violence against their partners (Medina, Schafer, Shear, & Armstrong, 2004). Therefore, there is no direct relationship between memory deficits and violence; rather, deficits in delayed recall could more likely be associated with deficits in executive functioning, which in turn could be more strongly related to the expression of violence (Krämer et al., 2011; Raaijmakers et al., 2008).

Attention

Several studies have revealed that cocaine abuse in adults is related to deficits in sustained attention and problems for fixating and shifting attentional focus (Spronk et al., 2013; Woicik et al., 2009). On the other hand, studies in children that were prenatally exposed to high levels of cocaine have revealed that, regardless of their sex and from age 3 to 7, they made more omissions in the *Visual Continuous Performance Test* than non-cocaine-exposed children (Bandstra, Morrow, Anthony, Accornero, & Fried, 2001). Furthermore, such deficits may be specifically circumscribed to the processing of visual information in the right hemisphere, as demonstrated by results from the *sustained visual orienting task* (SVOT) (Bandstra et al., 2001; Heffelfinger, Craft, & Shyken, 1997). Finally, such attention deficits may be caused by alterations in arousal regulation, affecting processing, learning and memorization (Heffelfinger, Craft, White, & Shyken, 2002).

Studies that have analyzed the relationship between attention deficits, cocaine and violence have focused on children and teenagers prenatally exposed to high levels of cocaine. Such studies concluded that children and teenagers with prenatal cocaine exposure tend to have greater attention deficits – especially in relation to sustained attention – and higher levels of externalizing behaviours (Bada et al., 2012; Carmody et al., 2011; Min et al., 2014). As is the case with memory, there is no direct relationship between attention and violence; rather, the relationship would be mediated by the executive functions (Tirapu-Ustárriz, Ríos-Lago, & Maestú-Unturbe, 2011).

Sex and concurrent use of alcohol and cocaine: precipitants and facilitators of violent behaviour

Sex is important on analyzing the effects of cocaine, since boys that were exposed to cocaine during gestation have more behaviour problems and cognitive deficits

(central processing of information, motor skills and abstract thinking) as they grow up than non-cocaine-exposed boys. Prenatally exposed girls, on the other hand, do not differ from non-exposed-girls in this respect. Moreover, the greater the exposure the more pronounced the behaviour problems and cognitive deficits (Delaney-Black et al., 2004). Comparisons between prenatally exposed boys and girls reveal that show more externalizing, risk and/or aggressive behaviour than girls (Allen et al., 2014; Carmody et al., 2011; Delaney-Black et al., 2000). Inhibitory control increases as the children grow older (from 7.5 to 11.5 years of age), even among boys; girls, however, have better prognosis as they improve earlier (Bridgett & Mayes, 2011; Carmody et al., 2011). Therefore, the neurotoxic effects of cocaine can cause more damage to the CNS in men than in women (Allen et al., 2014; Carmody et al., 2011; Chang, Ernst, Strickland, Mehringer, & Mark, 1999).

Generally, the relationship between the use and/or abuse of cocaine and violence is more evident in men than in women (Allen et al. 2014; Chermack et al., 2010), as men are more prone to being physically aggressive in general and against women in particular. In women, the aggression is verbal, and is usually directed at her offspring (Gómez et al., 2008). In both men and women the relationship between antisocial personality disorder and aggressive behaviour is clearer when there is abuse of substances, such as cocaine (Lewis, 2011; Mattson, O'Farrell, & Lofgreen, Cunningham & Murphy, 2012).

With regard to the concurrent use of cocaine and alcohol, the risk of displaying violent behaviour and an increase in violent thoughts is greater than that produced by the separate effects of each one of these substances (Chermack & Blow, 2002; Chérrez-Bermejo & Alás-Brun, 2014; Kraanen et al., 2014). This could be explained by the fact that the combination of the two substances can lead to the formation of a metabolite called *cocaethylene*, which inhibits the reuptake of dopamine in the systems of impulse control, such as the nucleus accumbens (Chermack & Blow, 2002; Soler-González, Balcells-Oliveró, & Gual-Solé, 2014). As stated earlier, neuropsychological deficits can play an important role in the facilitation of violent behaviour. Nonetheless, on comparing different cognitive domains in three groups of former users (cocaine, alcohol and alcohol-cocaine) there were statistically significant differences only between ex-cocaine users and ex-alcohol users. In fact, the group of ex-cocaine users showed less mental flexibility (assessed through the *Bexley-Maudsley Category Sorting Test*), poorer short-term/working memory for visual information and lower processing speed (assessed through the Sternberg Task and the Processing Speed Index [PSI] of the WAIS-R) than ex-alcohol users and consumers of both substances (Lawton-Craddock, Nixon & Tivis, 2003). Thus, the neuropsy-

chological deficits in people that abuse cocaine and alcohol and would not be as pronounced as in those that use cocaine exclusively.

In addition to these two factors, there are others that come into play, such as socio-economic status, which is a mediating factor in the relationship between cocaine abuse and violence. In general, people from the lower socio-economic status strata and those who are divorced, single or unemployed would be at greater risk of their cocaine abuse facilitating the expression of violence (Tar-diff, Marzuk, Leon, Portera, & Weiner, 1997).

Neuronal correlates

Neurotransmission

It has been hypothesized that cocaine facilitates aggression by inhibiting the reuptake of monoamines such as dopamine, noradrenaline, and serotonin or by overstimulating the postsynaptic receptors (Cunningham & Anastasio, 2014; Grewen et al., 2014; Moore et al., 2008; Patkar et al., 2003; 2006). Hence, the levels of monoamines in the synaptic space might increase (Cooper, Bloom, & Roth, 1991; Matuskey et al., 2014) in different areas of the prefrontal cortex and limbic system, which play a key role in the regulation of behaviour and emotions (Davis, 1996). Serotonin could be an important mediator in the relationship between the expression of violence and cocaine abuse (Patkar et al., 2006). Cocaine users presenting greater levels of violence show alterations in both the transporters of this neurotransmitter and in the postsynaptic receptors (Patkar et al., 2003; 2006). Such alterations in the monoaminergic system were corroborated by studies that analyzed the concentration of monoamine metabolites and monoamines in the cerebrospinal fluid of children with prenatal exposure to cocaine, concluding that these children presented higher levels of noradrenaline and its precursors and reductions in dopamine metabolites (Mayes et al., 1998; Needlman, Zuckerman, Anderson, Mirochnick, & Cohen, 1993).

Brain structures

Cocaine users have deficits in emotional decoding that might be due to the small size of their amygdala, as has been observed by some authors (Makris et al., 2004). In addition, these individuals might display less physiological activation, assessed through the amount of pupillary response, with regard to social interaction (Preller et al., 2014). Weaker activation of the medial orbitofrontal cortex could be a neural correlate of this kind of interpersonal interaction. Lower activation of this region would be associated with smaller social networks (Preller et al., 2014).

The high prevalence of impulsiveness and violence, as well as the greater reactivity and increased anger in

response to stress observed in briefly abstinent cocaine users may be linked to poor neurocognitive inhibitory control (Bell, Foxe, Ross, & Garavan, 2014; Fox et al., 2007; Fox, Hong, Siedlarz, & Sinha, 2008). Low activity of the following structures would be the neural correlate of such deficient control: the medial and inferior frontal gyri of the right hemisphere, the right inferior parietal lobe, the bilateral insula, the medial cingulate cortex and the supplementary motor cortex, whose hypoactivation could affect the regulation of behaviour through its interaction with the limbic regions (Bell et al., 2014; Pawliczek et al., 2013). Furthermore, these users also display low activation of the lateral orbitofrontal cortex and the ventral striatum, which is associated with greater anger expression (Goldstein et al., 2005), as well as poorer emotional recognition of anger (Calder, Keane, Lawrence, & Manes, 2004; Murphy et al., 2003). On the other hand, chronic cocaine users display less mental flexibility, which is linked to greater connectivity between the left medial frontal gyrus and the nucleus accumbens in periods of repose (Camchong et al., 2011).

In turn, cocaine-dependent adults have less grey matter and less activation of the anterior rostral cingulate cortex, and this would be subjacent to the low behavioural self-awareness and/or self-monitoring these people usually display (Moeller et al., 2014). All of this, together with the deficits in empathic processes, would affect decision-making processes, given the role they play as somatic markers of behaviour (Verdejo-Garcia & Bechara, 2009).

Lastly, children and teenagers (of both genders) exposed to high levels of cocaine have less grey matter and more cerebrospinal fluid in the prefrontal and frontal superior cortex (especially dorsal), the superior frontal gyrus, the precuneus and the parietal, limbic and paralimbic cortex than those not exposed to cocaine. Such structural deficits might explain their poor behaviour regulation and their sub-par executive functioning (Grewen et al., 2014; Rando, Chaplin, Potenza, Mayes, & Sinha, 2013). Furthermore, children aged 3-6 that were prenatally exposed to cocaine are less empathic to the crying of other babies and the discomfort of their mothers and show poorer capacity for cooperation, and this could be explained by a greater activation of the frontal right hemisphere (Moilanen et al., 2010). These results would be consistent with the deficits found in adult cocaine users that are prone to being violent.

Conclusions

The deficits described so far provide us with a deeper understanding of the perpetration of violence by cocaine users. The majority of studies have focused on deficits in empathy and executive functions, as these are important for social adaptation. Deficiencies in the decoding of

emotions (which could be explained by poor sustained attention capacity) may be subjacent to an inability or low ability to comprehend others' feelings and thoughts and for decision-making, as these people would not properly appreciate the consequences of their actions. Furthermore, it should be noted that the risk of violence is higher when one's abilities to verbalize emotions and think abstractly are severely altered. Thus, cocaine users may have difficulties for thinking logically, increasing the risk of their using violence, as they are unable to appropriately channel or express these internal states. Greater activation in different cortical regions of the right hemisphere regarding empathic processes might be considered as an indicator of greater right lateralization of emotional processing. However, the scientific literature to date claims that cortical processing of positive emotions is lateralized in the left hemisphere, whilst that of negative emotions is lateralized in the right. In consequence, this abnormal activation pattern characteristic of aggressive cocaine users would underlie the bias towards hostile processing of emotional information. Poor emotion regulation, whose neural correlate is a smaller amygdala than that of non-users, and poor inhibitory control and decision-making, defined by hypoactivation of different regions of the prefrontal cortex, could explain such excessively violent responses. Such deficits appear to be more evident in men, their effects at a neurological level having poorer prognosis than in the case of women. Despite that the concurrent use of cocaine with other substances, such as alcohol, would increase the risk of violent reactions, the related neuropsychological effects do not seem to be as marked as in other scenarios. An important limitation of the studies conducted to date is the variability of the populations used, since most of the studies have a very small sample size, and have considered different periods of abstinence, different socio-economic levels, different types of drug-use history. The present article, together with another addressing the neuropsychological profile of men convicted for intimate partner violence (Romero-Martínez & Moya-Albiol, 2013), highlights the importance of a certain homogeneity in the deficits presented by violent drug users. Thus, it would be of great utility to develop a single neuropsychological battery for the assessment of the propensity for violence in the absence or presence of drug abuse, adjusting it to a series of tests tailored to this population subgroup. This, together with the consideration of diverse psychobiological variables shown to be relevant in violent people (Romero-Martínez et al., 2013a; 2013b; Romero-Martínez, González-Bono, Lila, & Moya-Albiol, 2013; Romero-Martínez, Lila, Conchell, González-Bono, & Moya-Albiol, 2014; Romero-Martínez, Lila, Williams, González-Bono, & Moya-Albiol, 2013), would permit the development of intervention strategies that focus on these specific cognitive domains.

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