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"Bad genes" & criminal responsibility

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ABSTRACT

The genetics of the accused is trying to break into the courts. To date several candidate genes have been put forward and their links to antisocial behavior have been examined and documented with some consistency. In this paper, we focus on the so called "warrior gene", or the low-activity allele of the MAOA gene, which has been most consistently related to human behavior and specifically to violence and antisocial behavior. In preparing this paper we had two objectives. First, to summarize and analyze the current scientific evidence, in order to gain an in depth understanding of the state of the issue and determine whether a dominant line of generally accepted scientific knowledge in this field can be asserted. Second, to derive conclusions and put forward recommendations related to the use of genetic information, specifically the presence of the low-activity genotype of the MAOA gene, in modulation of criminal responsibility in European and US courts.

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1. Introduction

Based on current scientific evidence, it is now firmly accepted that both, the biological and psychological makeup of an individual, play an important role in the etiology of antisocial behavior and subsequently also in criminal behavior (Raine, 2013; Rhee & Waldman, 2011). Researchers have estimated that aggression, which is known to be linked to criminality (Buckholtz & Meyer-Lindenberg, 2008), is highly heritable and estimated that within any given community approximately 10% of families may be responsible for more than 50% of criminal offenses (e.g., Barnes, Beaver, & Boutwell, 2011; Farrington, Barnes, & Lambert, 1996; Ferguson, 2010; Rhee & Waldman, 2011). Furthermore, individual differences in antisocial behavior, are explained by both genetic and environmental factors, with genetics estimated to contribute about 50% of the variance, particularly in persistent antisocial behavior (e.g., Burt, 2009; Fergusson, Boden, Horwood, Miller, & Kennedy, 2011; Moffitt, 2005; Tuvblad, Narusyte, Grann, Sarnecki, & Lichtenstein, 2011). The proposition that genetic factors have a significant influence on antisocial behavior implies that there is a genetic propensity or predisposition in some individuals to engage in this type of behavior.

A person's genetic makeup (genotype) predefines a range, which determines the individual's potential and limitations. Environmental factors determine the exact location of the limitation within that range. For instance the genotype of an individual may predetermine that their intelligence (IQ) will be in the range between 90 and 110. It is that person's environment, which will determine the expressed IQ (phenotype) from within that range. However, even with optimal conditions the individual in question will not exceed the upper limit of 110 and attain for example an IQ of 130. Therefore, our genes (nature) shape our limits, predispose us in certain directions but do not lead us inexorably to any fixed outcomes.

In fact, as Raine (2008) and others (e.g., Baker, Bezdjian, & Raine, 2006; Tremblay & Szyf, 2010) point out, psychosocial influences can generate changes in gene function, these in turn result in modifications of neuronal functioning that influence behaviors. Moreover, the broad variability of individual behavior among individuals, who share the same genetic makeup, clearly demonstrates that genes do not play a deterministic role in predicting behaviors (Baker et al., 2006).

From a practical viewpoint, it is important to determine the implications of these findings for the criminal law and the legal system in general. It is imperative that legal and law enforcement professionals understand that human behavior (including behavior deemed criminal) is a complex phenomenon, which is influenced both by genetics and by the environment, and that genetic predisposition does not suggest determination, but a propensity, which is shaped by the environment. Further, it is also necessary to clarify the legal treatment in instances, in which a strong correlation has been supported between genes and antisocial behavior.

In this paper, we summarize the current scientific knowledge related to the links between genetics and behaviors as they relate to the monoamine oxidase A (MAOA) gene. We focus on this gene, because of the studies carried out on the genetic etiology of antisocial behavior, the research findings related to this gene are the most consistent. Moreover, evidence based on this gene has already been utilized in several trials with varied degrees of success.

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The interest in understanding the genetic etiology of criminal behavior, and the influence that it may have, and in particular the MAOA gene, is growing in the legal system, among academics, lawyers, judges, police, and law makers, as genetic profiles of the accused have began to break into the courts. While there are some who are proponents of these developments (e.g., Zeki & Goodenough, 2004), the majority of the legal academics and professionals in the area of neuroethics remain skeptical (e.g., Farahany & Coleman, 2006). Despite this debate, to our knowledge, there have already been two convictions in Europe, both in Italy (in 2009 and 2011; Bayout and Albertani, respectively) and one in the United States (State of Tennessee vs. D.B. Waldroup in 2009), in which the defendants' MAOA genetic profile was not only admitted as evidence, but also, and this is most important, have been used to support a mitigation of criminal responsibility.

While some consistent information related to the link between this gene and behavior is available, much still remains to be understood. Thus, these judgments may be premature, raise some alarms and highlight the current problematic issue of the use of neuroimaging and genetic profiles as evidence in courts (Jones, 2006; Jones, Buckholtz, Schall, & Marois, 2009; Morse, 2011a, 2011b; Nadelhoffer and Sinnott-Armstrong, 2012; The Royal Society, 2011; Yang et al., 2008; Farah et al. 2009). One of the key concerns is that the same genetic information is used to mitigate criminal responsibility and also to elevate perceived predetermined dangerousness and the resulting punishment.

Thus, the goals of this paper are twofold. First, we synthesize the current scientific knowledge regarding the low activity variant of the MAOA gene and its role in criminal behavior. Second, we extract the appropriate legal consequences and offer recommendations for the use of this information in legal proceedings.

2. MAOA-L allele and impulsive aggression in males

2.1. Monoamine oxidase A (MAOA) gene

The genes with the greatest demonstrated influence on antisocial behavior are those related to neurotransmission (Ferguson & Beaver, 2009). For this reason, the majority of scientific studies have focused on examining a set of *candidate genes*, associated with the breaking down of neurotransmitters, primarily dopamine (DAT1, DRD2, DRD4; Barnes & Jacobs, 2013) and serotonin (5-HTTLPR) or with the enzymes responsible for their degradation (COMT, MAOA; Cicchetti, Rogosch, & Thibodeau, 2012). Of these genes, research related to the monoamine oxidase A (MAOA) gene and antisocial behavior has produced the most consistent findings both in animals and in humans (e.g., Shih, Chen, & Ridd, 1999).

The MAOA gene is an enzyme located on the X chromosome. One of its key roles is to regulate neurotransmitters, such as serotonin and dopamine from the prenatal period throughout the life-course (Buckholtz & Meyer-Lindenberg, 2008). Given that males [XY] have only one X chromosome while females [XX] have two, females have two versions of the same gene carried on each of the X chromosomes. Thus, males are more vulnerable than females to any abnormality carried on this chromosome, as they lack a second version that could mitigate the influence of the affected gene, enhance its functionality or efficiency.

MAOA polymorphism consists of a variable number of tandem repeats; a nucleotide sequence of 30 repeated base pairs, which may contain 2, 3, 3.5, 4 or 5 repeats. Thus, the particular transcription of this gene region can occur in different versions and its variability depends on the number of repeats of a certain sequence of base pairs. Among Caucasians, for example, the most common genotypes are 3 or 4 repeats (3-rps or 4-rps), which are carried by approximately 30% or 55% of this population, respectively. The low activity MAOA genotype or allele (MAOA-L) is characterized by 2 or 3-rps, while the high activity MAOA (MAOA-H) is characterized by 3.5 or 4-rps. The 5-rp is associated with both low and high activity MAOA, but most commonly also to a low activity. These variations in the gene transcription have a strong impact on the efficiency of the enzyme and thus on the regulation of serotonin and dopamine (e.g., Beaver, Nedelec, Wilde, Lippoff, & Jackson, 2011). Due to this functionality, variations in the MAOA gene efficiency are highly relevant in the regulation of brain chemistry. Furthermore, as serotonin and dopamine are potent regulators of cognitive and behavioral functions, anything (including MAOA) altering or enhancing the serotonergic or dopaminergic systems has a great impact on the central nervous system and specifically on cognitive, behavioral and emotional regulation (Walsh & Bolen, 2012).

2.2. Aggressiveness and impulsivity: neurological and psychological phenotype of MAOA-L

2.2.1. MAOA-L as risk factor of aggression as a form of antisocial behavior

Brunner et al. (1993) examined several generations of a Dutch family, which had gained notoriety as all of its male members engaged in a pattern of behavior (phenotype) dominated by extreme and persistent reactive aggression. They were also identified as having a rare mutation of the MAOA gene, represented by 0-rp alleles, resulting in a functionally blocked gene (functional knockout). This extremely low enzyme efficiency was also linked to serotonin dysregulation.

In addition to this extreme case of completely functionally blocked MAOA, a link between serotonin dysregulation due to the low efficiency MAOA (or MAOA-L; 2 or 3-rps) and impulsivity/reactive aggression, as a type of antisocial behavior, has been documented in a number of studies (e.g., Carver, Johnson, & Joormann, 2008; Crockett, Clark, Tabibnia, Lieberman, & Robbins, 2008; Halperin et al., 2006; McDermott, Tingley, Cowden, Frazzetto, & Johnson, 2009; Verona, Joiner, Johnson, & Bender, 2006) albeit generally with small effect sizes and found most consistently in males (e.g., Beaver, DeLisi, Vaughn, & Barnes, 2010; Guo, Ou, Roettger, & Shih, 2008). Through its influence on the serotonergic system, it increases the vulnerability of the individual to antisocial traits or risk factors for antisocial behavior, namely low self-control or impulsivity, and negative emotionality. As a result, this variant of the MAOA gene is sometimes referred to as the "warrior gene" or "violence gene".

However, it is important to point out that although the positive link between MAOA-L and aggression is currently the predominant view, the effects are generally small and they do not suggest that this so called "warrior gene" is alone responsible for aggressive behavior (Buckholtz & Meyer-Lindenberg, 2008), as we explain below. In addition, while plenty of evidence supports his link, the findings are not consistent across all conducted studies (e.g., see recent meta-analyses: Duke, Bègue, Bell, & Eisenlohr-Moul, 2013; Tielbeek et al., 2012; Vassos, Collier, & Fazel, 2013).

2.2.2. Neurological and psychological phenotypes — impulsivity and emotion regulation

Despite these inconsistencies, the predominant view with respect to the link between MAOA-L and reactive aggression, and thus to antisocial behavior prevails. In fact, these inconsistencies foreshadow the complexity of the gene to behavior link. In recent years, researchers have strived to understand the mechanisms leading from genes to behavior (Raine, 2008) by identifying the pathways through which genetic predisposition may be expressed in subsequent behavior: the pathways from genotype to phenotype. Many attempts to identify direct links between specific genes and gene expression have failed (e.g., schizophrenia, cancer research; Collins, Kim, Sklar, O'Donovan, & Sullivan, 2012). Similarly, researchers were unable to identify a specific gene, a so called "criminal gene", which was once hoped to be identified as a marker of criminal behavior (Paige Harden, Quinn, & Tucker-Drob, 2012). The quest for direct gene–behavior links has ceased with the advancement in the field of genetics and the understanding that the pathways from genes to behavior are complex and elaborate.

As a result, in order to disentangle these complex paths through which genes influence human behavior, scientists have shifted their

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attention to describing the intermediate biological phenotype and the personality pattern associated with it, which in turn shapes the behavior manifested by the individual. Intermediate phenotypes, such as fMRI assessed functional or structural brain alterations, are quantitative biological markers of manifest behaviors. As genetic alterations exert an influence of a greater magnitude on intermediate phenotypes than on manifest behaviors or illness, they are also relatively easier to detect (Meyer-Lindenberg & Weinberger, 2006). Thus, in order to clarify the causal relation between a specific genotype and a specific behavior observed in an individual, researchers first try to identify the neurological patterns (structural or functional brain differences) that represent the intermediate expression of the underlying genotype. In turn, these structural and functional brain differences (intermediate phenotypes, or phenotype 1) are expressed in a specific psychological pattern representing personality characteristics (phenotype 2). Finally, these personality characteristics interact with the environment experienced by the individual throughout the lifespan, and all of these components interact with each other and their combined influence results in the final expression or external outcome: the individual's behavior.

In recent years, this concept has been extended to explain antisocial behavior, and consequently also criminal behavior (e.g., Raine, 2008; Raine & Yang, 2006). In short, criminal behavior includes specific antisocial behavior deemed as a criminal offense by the criminal law system. Thus, researchers have began to examine and try to identify a set of markers or intermediate phenotypes for antisocial behavior, such as sensation seeking, hyperactivity, lack of fear, low-self control, negative emotionality, callousness, lack of emotions, serotoninergic system impairment, testosterone deregulation and others. These and other biological and psychological markers have been also identified as risk factors for criminal behavior.

In the case of the MAOA-L genotype, specifically, the link to reactive antisocial behavior has been explained through a set of "somatic markers" or neurological biomarkers, suggesting structural and functional changes in the corticolimbic circuits, associated with emotional regulation and cognitive control (Buckholtz & Meyer-Lindenberg, 2008; Raine, 2008; Viding & Frith, 2006). Importantly, these structural and functional brain changes have also been found in a normal, nonviolent sample (Meyer-Lindenberg et al., 2006 described below), suggesting that these impairments alone do not explain the link between MAOA-L and antisocial behavior (perhaps with the exception of the extreme case of Brunner syndrome). However, as these brain alterations, for example through the use of fMRIs, could be presented in courts as evidence, it is important to recognize them and to define exactly their potential relevance and impact.

Meyer-Lindenberg et al. (2006), examined specific structural and functional brain changes in healthy and, more importantly, nonviolent individuals with a low (-L) versus high (-H) enzyme expression (or activity) of the MAOA gene. The main conclusion of this study was that the MAOA-L gene is effectively linked to an overactive amygdala as well as a number of impairments in the control systems, both of emotions (through changes in the prefrontal cortex) and cognitive processes (through changes in the cingulate gyrus). The table bellow summarizes the specific findings (*p*. 6270):

- Brain structure: *Both sexes*: Significant volume reductions (less gray matter) in cingulate gyrus and bilateral amygdala, particularly in anterior cingulate cortex. Also, approximately an 8% reduction in insula and hypothalamus. *In males*, also changes in the orbitofrontal volume (Brocca's Area increased by approx. 14%)
- Brain function: *Both sexes*: Hyper-reactivity in the amygdala during emotional arousal along with hypo-activity of the cingulated, orbitofrontal as well as insular cortices = suggesting a diminished reactivity of regulatory prefrontal regions.
- Amygdala–OFC connectivity: *Both sexes*: Less connectivity found in both, but particularly in males.
- Emotional memory: *Males only*: Increased reactivity in the left amygdala and hippocampal formation during retrieval of negatively balanced emotional material.
- Inhibitory control: *Males only*: Pronounced hypo-activity in the dorsal anterior-cingulate during response inhibition.

As these authors highlight, their results suggest that the MAOA-L allele, particularly in males, is linked to changes in the brain circuits responsible for the regulation of emotions, emotional memory, and cognitive control, all of which are related to impulsivity and reactive aggression. The brain differences result in certain neurological features of the individual personality (male, in particular), which lead to a specific psychological pattern/characteristics (phenotype 2). Specifically, the personality traits associated with the MAOA-L allele are: a higher reactivity to threat agents (harm avoidance), an increased tendency to experience anger, frustration and bitterness (angry hostility) and a reduced sensitivity to signals that trigger and maintain prosocial behavior (reward dependence; Buckholtz & Meyer-Lindenberg, 2008) as well as a greater tendency to inflict pain and punishment (McDermott et al., 2009).

In addition, two of the key risk factors for antisocial behavior, low emotionality and low self-control (e.g., Kuhn & Laird, 2013), are both strongly influenced by a poor functioning serotonergic system (e.g., Barnes & Jacobs, 2013; Beaver et al., 2009; Beaver et al., 2010). Specifically, serotonin has a key role in the functioning of the amygdala, which is responsible for the emotional charge of everyday experiences. Based on the emotional charge of our experiences, we then encode them as dangerous or not, which later shapes our behaviors/responses. In other words, based on whether we felt fear or thrill in similar situations in the past, we may avoid dangerous situations or seek them out (e.g., DeLisi, Umphress, & Vaughn, 2009).

In summary, there is some evidence that, particularly in males, the MAOA-L genotype presents a relevant risk factor for antisocial behavior and personality. It has been linked to deficits in the processing of emotions and deficits in responses to stressors and impulse control. From these features, which ultimately influence manifest behavior, a specific personality pattern emerges, which presents a notable risk for impulsive/reactive types of aggression and violence, particularly in males. However, it has also been recognized as a significant factor in the clinical course of other mental disorders (e.g., Brummett et al., 2008; Buckholtz & Meyer-Lindenberg, 2008), including anxiety and depression in females (Williams et al., 2009).

2.2.3. Preliminary conclusion: MAOA-L "small effect size" in antisocial behavior

Based on the findings outlined in the previous sections, three preliminary conclusions should be properly highlighted. These conclusions are particularly relevant for legal purposes, as their implication is to limit the reliance solely on the MAOA-L genotype as evidence in courts.

A) First, the MAOA-L gene variant influences the levels of individuals' impulsivity and emotional regulation, however, this in itself does not necessarily result in aggressive or antisocial behavior as an outcome. The MAOA-L allele alone is not a consistent predictor of antisocial behavior. Given the current research, neurological and psychological differences linked to MAOA-L (3- and 2-rps) alone are not pathological with regard to these behaviors. However, it is important to acknowledge that the MAOA-L genotype implies a genetic vulnerability, which generates neurological and psychological changes and alterations.

Studies that examined the impact of this gene specifically on antisocial behavior found only small statistically significant effects. This suggests that the MAOA-L allele explains only a small v of proportion of the variation in antisocial behavior and that there are other contributing factors; including other genes and environmental factors, for example, childhood maltreatment. Accordingly, the presence of the MAOA-L genotype alone should never be presented as evidence of a diminished criminal responsibility or dangerousness.

As mentioned above, this genotype can be observed, not only in individuals who have already committed a violent crime, thus exhibited an impulsive aggressive reaction to a negative

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stimulus, but also in individuals who are legally mentally healthy and (at least, for the moment) non-violent. Therefore, consistent with the current scientific knowledge, the presence of the MAOA-L genotype merely indicates a reactive–impulsive tendency, or a propensity to engage in violent behavior of reactive nature, but not the expression of the behavior itself.

Moreover, impulsivity or heightened sensitivity to stressful situations is a risk factor for antisocial behavior. However, it is important to point out that most of us may present with this 'dangerousness' trait to different degrees in specific circumstances. Even more importantly, this propensity is only one of many traits, which make each of us different and shape our personality and consequently what behaviors we display. In addition, not everyone with high levels of impulsivity engages in antisocial behaviors. As impulsivity alone represents a mere risk factor, and not causal factor, for antisocial behavior, it has little relevance for the criminal law. As we will describe below, it is necessary that a specific level of impulsivity and its impact on specific capabilities relevant for the Law are demonstrated for it to be considered as a mitigating factor of criminal responsibility. Also for determining criminal dangerousness it is necessary to demonstrate that the individual presents a risk for recidivism in this case due to his impulsivity.

Finally, this conclusion should be applied to both types of MAOA-L (3- and 2-rps) as they both present an increased risk for engaging in reactive aggression. While in most studies participants with 3- and 2-rp alleles have been pooled together to form one MAOA-L group, two recent studies have examined these two genotypes separately. They found that compared to individuals with 3-rp alleles, those with 2-rps were at a greater risk for engaging in serious acts of violent delinquency from adolescence to early adulthood (Guo et al., 2008), they also engaged in more antisocial behaviors, and had higher rates of arrests and incarcerations across the lifespan, however, they did not display more psychopathic traits (Beaver et al., 2013). The former study also revealed functional differences between these genotypes, showing that the 2-rp allele had the lowest level of promoter activity compared to the 3- and 4-rps.

Notably, consistent with previous reports related to MAOA-L, these patterns were more pronounced (Guo et al., 2008) or only studied (Beaver et al., 2013) in males. Moreover, the findings reported by Beaver et al. (2013) were based on a sample of African-American males due to the reported low prevalence of the 2-rps in their Caucasian sample and in this population overall (for other examples see also Nikulina, Widom, & Brzustowicz, 2012; Sabol, Hu, & Hamer, 1998). Thus, these patterns need to be further evaluated with larger samples of males representing different racial backgrounds (and possibly also females).

These studies provide preliminary support for the possibility that the 2-rp allele genotype may be a particularly important marker for antisocial behavior. It is plausible that individuals with this variant may have a more severe impact on the brain chemistry, and therefore, may result in more serious behavioral manifestations. However, this hypothesis remains to be tested. Thus, individuals with the 2-rp allele may require, with caution due to the preliminary nature of these studies, a different legal response.

B) The second conclusion to keep in mind is that the link between this genotype and impulsive–reactive aggression has only been established in males, not in females. Males are three times more likely than females (37% vs. 12%) to have this genotype. The remaining 88% of females are far less vulnerable than males, because they either have the MAOA-H genotype (42%) or they have a heterozygous genotype (42%; Buckholtz & Meyer-Lindenberg, 2008; Eme, 2013; Sjöberg et al., 2007). Although the structural and functional brain differences in both sexes with MAOA-L are broadly comparable, the resulting phenotype is different. Specifically, while research suggests that males with the MAOA-L variant have a propensity to impulsive/reactive aggression, the findings with respect to females are much less consistent (Nikulina et al., 2012). The specific factors, for instance hormones, that may interact with the gene to generate diverse patterns of personality/behavior, remain unclear and may contribute to the inconsistencies in the current findings.

Due to the vast inconsistencies in the findings related to the expression of MAOA-L in females, it is not possible to extend the same legal conclusions for both sexes and their treatment in criminal proceedings should be clearly differentiated. Specifically, as research suggests that the link between MAOA-L and impulsive–reactive aggression is a masculine profile, consistently, it should not be considered when the defendant/accused is a female, as was the case in the Albertani Trial.

C) Finally, the findings suggest a link between MAOA-L and impulsive/reactive types of aggression only. Thus, for legal purposes MAOA-L has no relevance in instrumental or premeditated types of violence, typical, for example, for psychopaths.

3. MAOA-L and childhood maltreatment interaction: "medium effect size" in relation to antisocial behavior

Consistent with what we just discussed, and as our goal was to derive legally relevant implications from the available scientific evidence relating MAOA-L to antisocial behavior, it may seem that we should conclude our paper here, stating that at this point there is simply not enough evidence to support any such implications. It may seem illogical to assert categorically that, according to the current scientific knowledge, by itself, this allele predisposes to antisocial behavior in a statistically small way and maintain, at the same time, that the dominant scientific point of view is establishing a relevant association. However, as will be explained in the present section, the research has more to say about this. Specifically, the particularity of this gene is that it is part of a significant gene by environment interaction (G × E) in relation to violent and antisocial behavior outcomes (Caspi et al., 2002; Fergusson, Boden, Horwood, Miller, & Kennedy, 2011; Kim-Cohen et al., 2006; Moffitt, 2005).

3.1. The definition of the gene × environment interaction

In the past few decades, researchers have focused on examining how genetic (nature) and environmental (nurture) factors influence each other in relation to behavioral outcomes. In the past few decades, this idea has been also introduced in research exploring antisocial behaviors. The most influential in this area of research has been work carried out by Moffitt (2005). This study was the first of several studies (e.g., Baker et al., 2006; Barnes et al., 2011; DeLisi & Piquero, 2011), which have focused on examining the interaction between genes and the experienced environment by an individual at different stages of their life and the influence of this interaction on their behavior. In general terms, a significant G × E interaction suggests that both of these factors are relevant in predicting an outcome (Dick, 2011). More specifically, it indicates that the two factors interact with each other (i.e., influence one another) in influencing the outcome of interest. In statistical terms, this is referred to as an interaction effect. It can occur in conjunction with, or in absence of, a main effect, in which one or both of the factors would or would not also have an independent contribution to predicting the outcome. Notably, with respect to antisocial behavior, the G × E interaction effect is much stronger than the influence of either of the two factors alone. This has been demonstrated in various studies involving monozygotic twins (e.g., Kim-Cohen et al., 2006; Moffitt, 2005), which revealed that genetic factors alone do not directly predict behavioral outcomes (no main effect), however, they do so in

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interaction with environmental risk factors (G × E effect). This pattern of findings has been identified for a variety of genes (e.g., Wright, Schnupp, Beaver, DeLisi, & Vaughn, 2012), including MAOA (Byrd & Manuck, 2013).

As we mentioned before, this research highlights that the current understanding of the gene/environment relation cannot be merely of "coexistence" but of "convivence". In other words, the genes "live" with the environment to shape our behavior and personality and not only "coexist" with it blindly and autonomously. The influence of environmental factors upon our brain and body as a whole, will result in changes in the phenotype throughout our lives, as is suggested by the notion of "brain plasticity" and even modification of our heritable genetics, according to "epigenetics" (Davidson & McEwen, 2012; Szyf, 2013; Twardosz & Lutzker, 2010).

From another point of view, Barnes and Boutwell (2012) argue that, if the G × E interaction is mainly responsible for setting the final expression of our genes into motion and thus realizing our phenotype, and since the environment changes throughout the lifespan, it seems also possible and reasonable to expect a genetic influence on the changes in behaviors. Thus, by exploring the genetic origins of behavior changes, new opportunities for treatment and rehabilitation may be opened.

3.2. MAOA-L × childhood maltreatment and antisocial behavior

The dominant scientific literature (see Byrd and Manuck (2013), for a recent meta-analysis) recognizes that the relations between the MAOA gene and antisocial behavior are established through the interaction between genetic and environmental factors. That is, this gene is a potent risk factor for antisocial behavior, but only when it interacts with other risk factors of environmental nature, characterized by adversity in the periods of life (prenatal period, infancy, adolescence), which are critical periods for brain development and configuration. Evidence derived from twin studies suggests that in childhood personality traits and individual differences in behaviors are most affected by genetic influences (Bartels et al., 2007). Moreover, genetic influences on antisocial behavior have been reported to increase and shared environmental influences decrease from childhood to adulthood (e.g., Meier, Slutske, Heath, & Martin, 2011).

Among all the potential environmental pathogens, which may interact with this MAOA-L allele, the one that has shown the clearest and most consistent findings is the experience of childhood maltreatment (abuse and neglect; e.g., Tikkanen et al., 2010). Consistent scientific findings show that early exposure to this type of stress induces several structural changes in the brain development (e.g., Hanson et al., 2010; Rinne-Albers, van der Wee, Lamers-Winkelman, & Vermeiren, 2013). The most significant structural changes are a greater amygdala volume and a reduced volume in certain sectors of the prefrontal cortex and hippocampus. These changes have relevant implications, particularly, because the connections between the amygdala and the prefrontal cortex are directly involved in emotion regulation and implicated in several psychopathologies with clear projections to antisocial behavior. Findings also suggest that the neural circuits related to social and emotional functioning are more likely to be shaped by experiences and that early experiences are key factors in preparing individuals for facing future adversity in life, in terms of vulnerability or resilience (Davidson & McEwen, 2012; DeLisi et al., 2009).

Consistent findings suggest that exposure to childhood maltreatment alone, has a small to medium effect on antisocial behavior. It fact, childhood maltreatment appears to be a universal risk: the sooner and more severe the child maltreatment is the more likely the individual is to experience behavioral or mental health problems including antisocial behavior (e.g., Haberstick et al., 2013; Tremblay & Szyf, 2010).

However, the combined effect of childhood maltreatment with the MAOA-L allele, presents a very significant risk factor for antisocial behavior representing medium to large effect sizes. This effect is generated, given that the MAOA-L allele increases the individual's response to exposure to an adverse environment. As Moffitt (2005) observed, 85% of the individuals with histories of severe maltreatment, along with this genotype, engaged in serious criminal behavior before the age 26. The authors concluded that childhood maltreatment more strongly predisposes to antisocial behavior in adulthood among those with a decreased MAOA functionality (MAOA-L), which is insufficient to offset the changes in the neurotransmission system induced by the maltreatment. The 12% of individuals presenting with such interaction were, responsible for 44% of the convictions for violent crimes. The outcome of antisocial behavior was found only in those individuals who had a low activity genotype. Among individuals who had a high activity MAOA allele (MAOA-H), the incidence of abuse-related antisocial behavior was much lower than in the MAOA-L group, thus suggesting that different variants of a given gene may act as a risk versus protective factor, that would promote resilience or resistance in adversity (Byrd & Manuck, 2013; Caspi et al., 2002; Fergusson, Boden, Horwood, Miller, & Kennedy, 2012; Nikulina et al., 2012).

The finding by Caspi et al. (2002) related to the effect of the MAOA-L by maltreatment interaction on antisocial behavior in boys has, been replicated in several studies (Fergusson et al., 2011; Kim-Cohen et al., 2006; Moffitt, 2005; Weder et al., 2009) although not all (e. g., Haberstick et al., 2013; Huizinga et al., 2006; Prichard, Mackinnon, Jorm, & Easteal, 2008). Several of these authors have suggested that the deficits in the neural systems responsible for emotion regulation and memory, may explain the effects of this interaction, as they are also crucial for the encoding, retrieving and extinguishing of negative emotional information elicited during experiences of maltreatment during childhood. These findings are consistent with another line of research mentioned above (Meyer-Lindenberg et al., 2006) linking brain structures involved in emotional regulation, specifically the amygdala and medial prefrontal and orbitofrontal cortices, to antisocial and violent behavior.

In conclusion, according to current scientific knowledge, carriers of the MAOA-L gene variant are highly responsive to their environment (positive or negative) and particularly to significant maltreatment from childhood. For this reason the MAOA-L gene variant is often referred to as one of the more "plastic" genes and not a mere vulnerability (Beaver et al., 2011; Belsky et al., 2009). The combination of these two factors presents an increased risk (greater propensity) towards aggressive and antisocial behavior in adulthood. However, according to the most recent meta-analysis (Byrd & Manuck, 2013) based on 27 studies, the MAOA by maltreatment interaction was robustly replicated in 20 male cohorts, but not in any of the 11 female cohorts. Notably, in females, maltreatment had a main effect on antisocial and aggressive behavior. In a recent study of adolescent boys and girls, researchers (Aslund et al., 2011) found that maltreated boys with a MAOA-L but maltreated girls with one or two MAOA-H variants, where at a higher risk for delinquency. Thus, it is important to highlight two points here. First, that not all the studies have supported such interaction, confirming the moderating role of the MAOA-L (e.g., Byrd & Manuck, 2013; Haberstick et al., 2013) and, second, that this pattern of findings has only been documented in males. With regard to females, findings remain unclear and contradictory (e.g., Fergusson et al., 2011).

4. Legal implications

Inevitably, when it comes to analyzing the impact that genetics, as well as neuroscience, may have in criminal law, old and well-known prejudices emerge into the debate highlighting the fears of placing blame onto the biology of individuals and thus relieving them of responsibility. This, more or less subconsciously, pushes us almost immediately to reject this potential association (Zeki & Goodenough, 2006). Objectively, genetic findings, as well as the use of neuroimaging in courts, their potential uses and misuses, of course, bring up many ethical concerns, compelling us to be particularly cautious in their use (e. g., Gillett & Tamatea, 2012; Jones, Wagner, Faigman, & Raichle, 2013).

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However, the openness to consider the influence that genetics exerts on who we are as individuals, or its influence on shaping our experiences and behavior, does not imply that we also accept a deterministic and eugenic approach to criminal law, which is not endorsed or advocated by current scientific knowledge. In fact, as we tried to point out in the sections above, the more we know about the genetics of behavior, the clearer is the role of environment in shaping this link. In shaping the individual, the environment is intertwined with genetics, in a biology/environment interaction, which is close to impossible to split (e.g., Baker et al., 2006).

Moreover, in our opinion, one of the basic conclusions of this paper is to highlight the underestimation of some environmental factors by the Criminal Justice System. This shortcoming is particularly striking when it comes to the lack of consideration of maltreatment and/or severe adversity from infancy to late adolescence, when overwhelming evidence suggests that these experiences increase the risk of offending and mental health problems (e.g., Mersky, Topitzes, & Reynolds, 2011; Stewart, Livingston, & Dennison, 2008; Teicher, Anderson, Ohashi, & Polcari, 2013; Topitzes, Mersky, & Reynolds, 2012). Criminal law, in light of these scientific findings, and guided by the principle of equality, should respond to what science is telling us. At the very least, the Law should consider this issue openly and define a proper legal treatment of these circumstances, which is not adequately appreciated today.

However, returning to our discussion of genes, it is important to remember that, clearly, predisposition is not determination (Goodenough & Tucker, 2010). Moreover, as Jones (2006) pointed out, affirming that individual behavior is genetically influenced does not require, necessarily, denying that it should be viewed with a legitimate moral reproach nor mandate that every case receives mitigation in sentencing. As in any other case, the decision about the legal treatment of the MAOA gene will be a normative judgment. It should be made based on what science suggests but, essentially, based on a balance ensuring the protection of the society and respect for the constitutional guaranties and human rights of the individual.

Further, building on the philosophical theories of Moore and Hume, Jones (2006) suggests that when the potential implications of behavioral genetics in criminal law are analyzed, it should be remembered that "is" is not equivalent to "ought". In other words, it is not possible to extrapolate how things ought to be (or what is moral/right), from the fact that they occur. Thus, even if we were able to identify, with reasonable specificity, that a particular combination of alleles in a specific environment significantly increases the risk of committing certain criminal behavior, it is a different issue whether such information should or 'ought' to play any role in the Criminal Justice System. Explaining, thus, is not justifying; as well as describing is not prescribing. Normative conclusions cannot be directly derived from these facts, as it was proposed earlier and now represents a traditional mistake, the so-called "naturalistic fallacy". Even if we were to recognize such relevance for the criminal law, it would not be prejudged which should be the specific legal response. Thus, all decisions must be passed through a normative, moral and consequentialistic, filter. Genetic information could lead to solutions in criminal policy (and in fact, it does), aimed at different directions at the same time. The solution is always normative, for a specific time and society.

Thus, in our view, the legal treatment of MAOA-L genotype should be just equal to other analogous mental impairments of different or still unknown origin. Of course, *lege ferenda*, if after our analysis it may be considered necessary, reform proposals will be suggested. Therefore, our next step is to analyze whether the presence of MAOA-L may be considered under any of the defenses described in Art. 20 of the Spanish Penal Code (SPC), specifically, under the mental insanity defense stated in §1. In addition, we will explore its potential relevance in considering mitigating circumstances set forth in Art. 21 of the SPC and, particularly, in Art. 21.3 related to circumstance of provocation or fury.

Thus, we ask whether the presence of the MAOA-L allele, by itself, or as it is already more plausible, in interaction with an experience of significant maltreatment in the early stages of life, may lead to some "defect of reason" and thus have an impact on the defendant's liability, in accordance with the definition of accountability. If this was the case and depending on the severity of the impact on one's judgment, it would be necessary to recognize it as substantiation of an insanity defense or mitigation based on the same grounds. Thus, contrary to Farahany and Coleman (2006), we do not contend, at least under the Continental legal system, that only because accountability is defined by the law based on an objective standard of a person, it would not be possible to concede any relevance of the genetic profile of the defendant. Even though the law starts with a presumption of accountability as an objective standard, in fact, it questions the specific defendant's mental capacity.

Under the Spanish criminal law, for example, three outcomes would be possible in case of impairments in the defendant's mental state. A) First, an insanity defense ("eximente completa de anomalía o alteración psiquica"), when the defendant is found to be completely irresponsible for the offense (a non-rational agent in the moment of the crime), presenting a fully or severely abnormal state of mind, which makes him inaccessible to the criminal rules. Thus, the defendant is not able to know what he is doing, distinguish right from wrong, understand the illicit meaning of the act (the "nature and quality" of the act and that it was wrong); and/or he lacks the capacity to control his criminal impulses completely. The defendant, in such case, is not held responsible but he can be sent to a psychiatric institution. B) Second, also under a criminal rationality defect, a partial or incomplete insanity defense ("eximente incompleta de anomalía o alteración psíquica") when the defendant is found to have a significantly diminished responsibility that does not, however, warrant a full insanity defense. In this case, generally, the individual is subjected to a mitigated punishment in sentencing as well as treatment resources ("security measures"). C) Third, a mitigation ("atenuante") in sentencing (possibly also some security measures), when the defendant exhibits some, but not very relevant, impairments in his cognitive or control capabilities.

In the following sections, we explore these legal options in greater detail.

4.1. Potential inclusion of MAOA-L in the mental insanity defense

There are some differences in the approach to criminal capacity in the Continental vs. Common Law legal systems. We will outline both below.

4.1.1. Continental legal system

Article 20, §1 of the SPC, addresses the issue of the mental insanity defense and indirectly defines accountability or the capacity for criminal responsibility, as follows: "The following persons shall not be criminally accountable: Those who, at the time of committing a crime, due to any mental anomaly or alteration, cannot comprehend the unlawful nature of the act, or to act in line with that comprehension. A temporary mental disorder shall not result in exoneration from the punishment when provoked by the subject in order to commit the offence, or when he would or should have foreseen that it would be committed". This regulation is representative of Continental Law systems with regard to accountability. In fact, this definition is essentially coincidental to the one established in paragraph §20 of the German Criminal Code (Strafgesetzbuch — StGB), §11 of the Austrian Penal Code (StGB-Österreich), and very similar are also the provisions in the French Penal Code, Art 122-1, among others.

According to these legal descriptions, in order to be held criminally responsible, the defendant must have had two core capabilities, occurring simultaneously and completely at the time of the offense. First, is the cognitive ability or capacity to appreciate that the act is against the law, and thus wrong. At the same time, the defendant must also have an average capacity to conform his behavior to the requirements of the law (volitional, control or self-determination). The capacity for criminal responsibility, thus, ultimately is defined in terms of the

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defendant's ability to be motivated by the criminal law, which is the primary tool to prevent the commission of crimes (general prevention) in consequentialist systems.

4.1.2. Common Law system

In Common Law systems, in the United States (US) and England, the criteria for criminal liability are only broadly coincidental with the Continental system. The insanity defense, per se, is not recognized in three states of the US (Arizona, Utah and Montana). In the states where it is recognized the requirements for accountability differ from those mentioned above.

The grounds for an insanity defense are based on a lack of rational capacity at the time of the commission of the act alleged to constitute a criminal offense, due to cognitive impairments. It is necessary to prove that the defendant, at the time of committing the crime, suffered a defect of the mind, which prevented him to be aware of the nature and quality of his actions (to know what he was doing, without delusion) or he lacked the ability to distinguish right from wrong. In a successful insanity defense, the individual is sent to a mental health institution for an indefinite commitment, sentenced to prison for incarceration or both, depending on the specific legislature of the governing jurisdiction (e.g., Farahany & Coleman, 2006). In England a successful plea of insanity will lead, depending on the discretional decision of the judge, to hospitalization, supervision or an absolute discharge. As a partial defense, in a charge of murder, a defense of "diminished responsibility" is also possible, leading to a lesser charge of manslaughter. Under The Coroners and Justice Act 2001, this circumstance is grounded on a substantial impairment of the defendant's mental capacity, such that it prevents him to understand the nature of his own conduct, to form a rational judgment or to exercise self-control (Ormerod, 2011).

According to the rules known as the M'Naghten Rules, in a plea for an insanity defense, in order to establish whether the defendant knew what he was doing, at the time of committing the criminal act: "the jurors ought to be told in all cases that every man is to be presumed to be sane, and to possess a sufficient degree of reason to be responsible for his crimes, until the contrary be proved to their satisfaction; and that to establish a defense on the ground of insanity, it must be clearly proved that, at the time of the committing of the act, the accused party was laboring under such a defect of reason, from disease of the mind, as not to know the nature and quality of the act he was doing; or, if he did know it, that he did not know he was doing what was wrong" (M'Naghten's Case (1843) 4 St Tr NS 847).

Thus, the main difference between these two legal systems, regarding the mental insanity defense, is that under the Common Law system this defense is based exclusively on cognitive impairments. It does not require any tests to assess potential deficits in impulse control, in order to identify a lack of ability to refrain from engagement in illegal behavior due to volitional impairments (Donohue, Arya, Fitch, & Hammen, 2008). Historically, many States of the US accepted the recommendations of the Model Penal Code from 1962, which included consideration of both cognitive and volitional impairments and their assessment (ALI, §4.01, 1985). However, after the attempted assassination of R. Reagan in 1981, the consideration of the volitional impairment prompted a debate and became controversial to introduce in Courts. Finally, it was abolished in many states where it used to be prescriptive prior to this event (e.g., Aharoni, Funk, Sinnott-Armstrong, & Gazzaniga, 2008).

In the sections below, we describe the requirements of the SPC for establishing accountability and explore the MAOA-L/maltreatment as possible circumstance to consider in the mental insanity defense. We highlight in advance that the conclusion will be negative, but we consider it necessary to discuss, because any potential mitigation should be founded on the core elements underlying the insanity defense.

4.1.2.1. The time of assessment of accountability. According to the law, lack of accountability shall be proved at the time of committing the crime, unless the defendant put himself into the situation intentionally or with negligence.

4.1.2.2. Biological requirement: mental anomaly or alteration. The SPC requires that the lack of accountability or capacity for culpability is due to a psychiatric or mental anomaly or impairment. This is the so-called "biological element" of the insanity defense. This concept is much broader than the previous legal requirement of a diagnosable "mental illness" or "mental disorder", which is still in use in other systems. The latter requirement reduces the applicability of this circumstance to the presence of a mental disorder recognized in a diagnostic classification manual, for example, the DSM-5 (in US, American Psychological Association, 2013) or ICD (in Europe, WHO, 1993). Such a requirement would directly exclude the application of exoneration based on Art. 20.1 of the SPC due to this circumstance, as it is the case in other legal systems requiring a diagnosable mental disease per se.

The different levels of MAOA activity have been associated with different risk factors and with different mental disorders, for example: depression and antisocial personality disorder. The "*aggressive–reactive impulsivity*", documented in those with the MAOA-L genotype, may therefore be a symptom of such disorders, but, strictly speaking, impulsivity is not a mental illness and thus MAOA-L does not directly indicate the presence of a diagnosable mental disorder.

Therefore, legal systems, which build the insanity defense on the grounds of a diagnosable mental disorder, as Common Law does, would exclude, ab initio, the potential application of the insanity defense in cases where the crime may have been committed as a result of MAOA-L/maltreatment dysregulation. In these normative systems, the impulsivity impairment related to this circumstance could only be considered, as another relevant factor, based on the hypothesis of comorbidity with any other documented mental disorder (e.g., psychosis), in evaluating the overall impact of the different impairments on the defendant's mind and judgment. This is currently common practice in cases where the accused exhibits several concurrent elements, which are relevant to exonerate or reduce their criminal responsibility (e.g., a psychotic disorder along with drug or alcohol abuse/dependence).

In SPC, however, as in other Continental systems, the legal text is significantly broader. The concept of abnormality or mental alteration has a wider meaning and it allows, based on its wording, any pathological brain deficiency, either structural or functional, as well as any other mental impairment without a documented brain damage or dysfunction, as might be for example evident in the case of post-traumatic stress syndrome. Thus, what matters, is that the individual's psychological functioning, for any reason of biological nature, is anomalous, pathological, and deficient compared to an average individual from the normal population.

Accordingly, in our view, the presence of the MAOA-L/maltreatment could, enter the sphere of the insanity defense, because it involves a mental alteration with clear physiological correlates: It is linked to a poor performance of the serotonergic system, and alterations in the structural and functional characteristics of the brain as well as compromised connectivity. Moreover, little doubt remains about the psychological or psychiatric nature of this alteration. It makes no real sense to make distinctions between the "brain" and the "mind", as they are both an indivisible whole (Damasio, 1994). Thus, brain impairments are also mental impairments, which are easily appreciated regarding the psychological pattern associated with the MAOA-L. The physiological alterations related to MAOA-L affect the neural circuits of emotional regulation and moral decision-making, thus also the personality and behavior of individuals. Ultimately, it is a mental alteration with a genetic and environmental etiology. In fact, the newest revision of the DSM (DSM-5, APA, 2013) acknowledges that conduct or behavioral problems are rooted in problems of impulse control and affect regulation.

This conclusion, however, may be considered striking regarding a genotype, of which one of the low variants – the 3-rp allele variant – is relatively common among Caucasians as it is carried by approximately

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30% of the population of this race. However, in our opinion, to the extent that this allele determines a malfunction (below average functioning) of the neurotransmission system as described above, it constitutes a mental alteration. Another issue, which also needs to be clarified, is the specific legal consequences that must eventually ensue from this mental alteration. As mentioned above, preliminary evidence suggests that the 2-rp allele may be a particularly relevant marker of antisocial behavior, therefore this variant should be treated differently (if with caution at this point) than the 3-rps. Moreover, there should be no doubt about the pathological nature and effects of the more rare mutations that result in Brunner syndrome (0-rp). The 5-rp variant of the genotype, to the extent that science consistently may associate it with abnormal regulation of the serotonin system, should be subject to the same treatment as individuals with the 3-rp allele.

Finally, it is necessary to point out that the dysfunctions associated with the MAOA-L cannot be generalized to all situations. Specifically, they are limited to a greater vulnerability to triggering events and the individual's propensity to respond impulsively, with aggression to such provocation. The offense has to correspond to this behavioral pattern and has to be "functional", involving "reactive aggression" (Blair, 2001, 2004, 2010). Even in cases of extremely violent crimes, it should not be considered when violence was used instrumentally (i.e., for a specific gain), as it is characteristic of psychopaths, for instance, since the effects that the MAOA-L generate are not related to this type of goal-oriented violence (Blair, Mitchell, & Blair, 2005). Accordingly, the MAOA-L profile could not be taken into account to argue insanity, for example, in a money laundering offense, fraud, rape and in a peculation.

4.1.2.3. Normative element: "cannot comprehend the unlawful nature of the act, or to act in line with that comprehension"

4.1.2.3.1. MAOA-L/maltreatment and the impairment of volitional capacity. As we mentioned before, research clearly suggests that it is impossible to separate our cognitions and emotions, to the extent that both aspects of the human mind are intertwined, interacting in a wide range of psychological processes, including the decision making process. Research has not established an association between the MAOA-L and cognitive deficits, with the exception of mild mental retardation related to Brunner syndrome. Furthermore, no correlation has been established between any cognitive deficits and the gene/environment interaction. Thus, to start with, we have to rule out that the MAOA-L/maltreatment interaction may be related to any impairment of the defendant's cognitive capacity under Art. 20.1 SPC. In other words, this interaction does not influence the defendant's ability to appreciate what he is doing and the illicit nature of his actions. However, we must consider whether in these cases the individual's volitional capacity may be influenced such that their motivational skills, which would enable (as any average person) avoidance of the illicit behavior and choice of a licit one, may be compromised. The defendant, in order to be considered accountable, has to be able to "understand" that what he is doing is unlawful and, at the same time, he has to be able to control his illicit impulses, and have an average capacity to act in line with that comprehension.

As we discussed at the beginning of this work, the antisocial behavioral pattern related to the MAOA-L/maltreatment interaction is impulsive and reactive aggression. Individuals with this genotype facing a triggering or provocative event, are much more easily irritable, frustrated and reactively aggressive. Therefore, under these specific circumstances, their capacity for self-control is affected, as a result they are more impulsive and, thus, have greater difficulties to adapt their behavior to the requirements of the Law; to control their impulses for illicit conduct and adapt their behavior to the legally binding prosocial behavior. In conclusion, the impact of the MAOA-L/maltreatment on the defendants' accountability is through brain alteration, which affects their capacity for effective self-control.

However, this statement must be immediately qualified, by pointing out that this association is relevant to the criminal law only as far as, in addition to the MAOA-L variant, a history of childhood maltreatment in the defendant can also be documented. It is the interaction of the two that influences the individual's self-control and thus also accountability. By itself, the MAOA-L is linked to brain impairments, with functional and structural changes, however, it has not been consistently linked to a pathological pattern. These brain changes alone, even at the presence of a consistent association with higher impulsivity, represent mere individual differences, which should not receive any specific legal treatment. In other words, based on the sole presence of MAOA-L, exoneration (or insanity defense) should not be considered for a full or partial defense. It should also alone not be considered as a mitigating factor in sentencing.

Thus, the only relevant impact on the individual's accountability will be considered in individuals, who are carriers of MAOA-L and have also suffered severe abuse (physical, sexual, psychological) or neglect early in life, in the period from birth to early adolescence. In order to meet criteria for the impairment of volitional capacity, both of these circumstances must be reliably proven in the legal process, along with the nature of aggression as reactive and linked to impulsivity.

At this point we must note again, although we do not have the space to discuss this concern at length in this paper, that the use of neuroimaging as well as genetic evidence in courts are both still largely debated topics. Among others, what is perhaps the most remarkable is the discussion about what exactly can be proven and to what degree by these scientific tools/approaches. Science is challenged to provide evidence of ontological facts in the backdrop of normative rules, while also respecting the stated rules of evidence. In the case of MAOA-L discussed here, behavioral genetics and neuroscience are called to prove cognitive impairments specifically regarding the unlawfulness of the defendant's behavior and/or volitional impairments related to the defendant's capacity to avoid the antisocial (criminal) behavior. In addition, the law also requires that the strength or level of these impairments in the moment of committing the crime is proven (Aharoni et al., 2008; Morse, 2011b, 2011c; The Royal Society, 2011). However, notably, these are also difficult to prove through currently used tools (psychological and clinical) in courts.

4.1.2.3.2. Lack of consistent scientific findings regarding the strength of impact: exclusion of the insanity defense. The most problematic issue regarding the legal treatment of the MAOA-L/maltreatment interaction is to determine the specific strength of its impact on the capacity for self-control. It is not sufficient to prove the biological element of the defense in support of the individual's deficient volitional capacity. In order to apply a full or even partial exoneration, or any mitigation of the punishment, the relevance or strength of impact on the mental/behavioral functioning of the individual must also be considered. The full defense requires a complete lack of the individual's volitional capacity, while a partial defense (Art. 21.1 regarding art. 20.1 SPC) necessitates a very severe or very relevant impairment. Less severe or less significant impairments only allow the application of mitigating circumstances under Art. 21 SPC.

Based on our review of the scientific literature, there is no available data from which clear conclusions could be drawn with respect to the severity of the impact on individuals' volitional capacity. Not surprisingly, there are also no findings suggesting a total lack of capacity for self-control. For these reasons, we must completely rule out the consideration of an insanity defense in these cases, leaving only the possibility of using this information for mitigation in sentencing. Thus, legally, the defendant shall be found "guilty", without impairment in his legal accountability strictly speaking, but with grounds for mitigation.

In short, from this analysis we should retain the following considerations: 1. MAOA-L implies a mental alteration, pathological if a MAOA-L/maltreatment interaction can be documented; 2. this interaction affects volitional capacity; 3. science has not yet supported a severe impact, thus, legally, it will only permit a mitigation; and 4. such potential mitigation has to be founded on the grounds outlined in points one and two.

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4.2. Mitigation under art 21.7 and the circumstance of "fury" (provocation) under art. 21.3 SPC

From here, the solution depends on the possibilities offered by each legal system. In the Spanish Law, art. 21 SPC lists several mitigating circumstances and also, an analog final general clause, which permits the consideration of other circumstance that may provide the same grounds for a lighter sentence as the ones explicitly listed (art. 21.7ª SPC). For example, with respect to non-violent offenses resulting from a compulsive need to profit (e.g., stealing), this vulnerability that weakens the individual's self-control, is often taken into account in mitigation, based on the provision of 'other circumstances' allowed to be considered. Consistently, the MAOA-L/maltreatment interaction may be easily located and considered under this article.

In addition, the SPC offers another option, which is to apply the circumstance stated in art. 21.3ª that outlines cases, in which "the defendant acted due to causes or stimuli so overpowering that they produce fury, obstinacy or another similar emotional state of mind", the so called provocation or fury circumstance. In essence, the application of this mitigation by courts requires two main elements: a cause and an effect or outcome, that are linked by a causal connection and, in turn, causally linked with a violent behavioral reaction of the individual; the prosecuted criminal conduct [see for example: STS de 19 de diciembre de 2002 (RJ 2003/321) or STS de 19 de octubre de 2006 (RJ 2006/7706), for all].

The cause is the triggering event or stimulus, which must be external to the individual and relevant enough based on social evaluation. The stimulus must be of such relevance or strength that it permits the explanation (not justification) of the criminal reaction that occurred. Originally, it was often required that the external stimulus comes from a preceding behavior of the victim (or target of the crime), however, such exigency has been nuanced in later sentences. The trigger should be, if not understandable, at least not socially reprehensible, for example, killing or injuring someone due to jealousy or a 'macho' attitude. Notably, regarding this, in the US and the UK the application of the provocation circumstance in cases of gender violence is currently under a heated debate (Fitz-Gibbon, 2013; Tilleman, 2010; Torry, 2001).

The effect is the individual's mental impairment, as outcome of the triggering or provoking event. A specific change in the mood of the individual must occur, such that it diminishes his accountability, or "a strong emotional reaction of fury, which decreases the will and intelligence of the defendant" (STS October 6, 2000, RJ 2000/9511). However, the mood change may not be so intense that a transitory mental disorder may ensue, as this would lead to a plea for a full or partial insanity defense. At the same time, the mood change shall not be so small that it would not exceed mere feelings of anger, flushing or mild lightheadedness. Although these milder emotional reactions often accompany some passionate offenses, they are considered legally irrelevant. Similarly, the mitigation should be denied when, objectively, the provoking stimulus is trivial and any reasonable adult person would respond to them without violence or aggression. Therefore, proportionality is required between the stimulus and the reaction of the individual.

Furthermore, depending on the particular characteristics of the effect, we must distinguish between "fury", "blindness" and a similar passionate state. Generally, an individual is said to have experienced "fury" when the reaction was a burst of emotion, which was momentary and swift, immediately following the stimulus; while "blindness" or "being blind with rage" has a longer duration and allows for a longer period of time between the stimulus and the response. Moreover, this nuanced jurisprudence allows for the possibility that this passionate state arises in response to a string of accumulated events, which occur along a period of time. In this case, the emotion remains latent until an explosion occurs ("the cup overflows"), due to a particular stimulus, which is, importantly, affected by the previous experiences.

In this case, the sudden emotional state, as well as the causal link between the triggering event and such state should be acknowledged, supporting that the behavior is a direct consequence of the importance of the stimulus. This has to occur in a reasonable unity of action, because the outburst or "fury" cannot be appreciated, if a reasonable period of time has past between the stimulus and reaction, which would permit the individual's recovery and calming of extreme mood.

Based on this understanding, we propose that this mitigating circumstance can be applied in the case discussed in this paper. Specifically, given that the MAOA-L/maltreatment interaction generates a significant impairment in an individual's capacity for self-control, due to a marked impulsivity, affecting inhibitions that would motivate him to refrain from antisocial behavior; and, given that the inherent risk of this interaction is a propensity for antisocial behavior, as an aggressive response to a triggering event, we suggest that: this interaction should be considered a mitigation in sentencing on the grounds of impairment of the capacity for self-control, and the mitigating circumstance of *fury*, as stated in art. 21.3ª SPC, should be applied.

Considering the requirements of the law, in applying this mitigating circumstance, the following should be considered:

- This subjective vulnerability must be appreciated as more relevant in cases, in which the requirement of the provoking event and the proportionality between it and the reaction or effect on the individual's mind can be clearly ascertained.
- The element of aggressive and impulsive reaction (reactive aggression) shall be documented in accordance with general requirements and in support of the individual's capacity for self-control having been affected and, therefore, although mildly, his accountability.
- The severity of the triggering event shall be evaluated in accordance with the social criteria of assessment. An event shall be deemed important enough, so as it might "explain" the angry reaction. However, the impairment in self-control could partially compensate for the lower social relevance of the event and/or the excess in the reaction. Of course for the individual, the event has to be subjectively significant.
- The higher the genetic vulnerability (i.e., MAOA-L 2rps or the concurrence with other adverse factors "toxic lives") and the more severe the situation of abuse or of early adversity, the stronger will be the ground for mitigation.
- Given the current scientific evidence, this specific circumstance and mitigation should be considered only in males.

In a situation with a triggering event, indeed, the illegal reaction of the individual is motivated partially (or amplified significantly) by a genetically based vulnerability, which does not need to exist conceptually for invoking the mitigation under art. 21.3 SPC. However, in our view, when the law refers to "fury, obstinacy or any other similar state of mind", it is referring to psychological states that promote and explain the wrongful behavior, without any prejudice about their origin. So, a genetic and an environmental factor, interacting with the external provocating event, could be included in the wording of the Law. The meaning of this circumstance is not affected only because the reasons for the individual's passionate reaction also have a biological component. Moreover, in our view, it is also important to remember that, today, from a scientific point of view, a purely rational reaction/response is not sustainable, either in favor of prosocial or antisocial behavior. In a "passionate" state of mind, as this circumstance describes, it is clear that both brain circuits of emotion and reason intertwine and clearly, the decision-making process involves both emotional and rational elements, which are crucially linked.

Science, however, as noted by Baum (2011), still has to clarify the nature of environmental factors that are relevant in this interaction. Questions related to the timing and severity of experiences need to be addressed in order to provide a more precise definition for "adversity in early periods of life" or "early and severe maltreatment". For example, it is not yet clear whether only abuse, which occurs within the family environment or also other types of abuse (such as bullying, exposure

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to gang violence), should be considered. Similarly with respect to neglect, should it be considered only when it occurs in the form of maternal rejection or also through other experiences, such as social and cultural exclusion. In addition, the co-influence of other negative life factors (e.g., comorbid illness, maternal smoking during pregnancy) may also need to be considered. In general, depending on the consistency of the expert evidence, all the influencing negative circumstances and life events of the accused may be considered in a potentially stronger mitigation.

The legal solution proposed here, in our opinion, can be understood as materially equivalent to that suggested by Baum for the Common Law system, although his was more tentative and limited in efficacy. Specifically, Baum (2011) proposed that, in case the current debate will lead to the subjectivity of the standard of "reasonable man", the partial defense of provocation could be invoked in MAOA-L/maltreatment cases.

Both, in the US and in the UK, the provocation defense is common. It is not a general defense, but a special defense to a charge of murder. Its consideration implies the recognition of a diminished responsibility such that if provocation is documented, it requires the reduction of the charge from murder to manslaughter, and accordingly an adjustment of the punishment.

The test of this circumstance requires answering the questions whether the defendant actually lost his control and whether a "reasonable man", under the same circumstances as the defendant would have reacted in the same way. The US Model Penal Code describes this situation in broader terms than the traditional configuration of this circumstance. Specifically, "murder" becomes "manslaughter" if the murder is "committed under the influence of extreme emotional disturbance for which there is reasonable explanation or excuse. The reasonableness of such explanation or excuse shall be determined from the viewpoint of a person in the actor's situation under the circumstances as he believes them to be" (section 210.3(I) b). In the English criminal law, under The Coroners and Justice Act 2009, ss 54–56, provocation is defined as follows: "(1) Where a person (D) kills or is a party to the killing of another (V), D is not to be convicted of murder if — a) D's acts and omissions in doing or being a party to the killing resulted from D's loss of self-control, b) the loss of self-control had a qualifying trigger, and c) a person of D's sex and age, with a normal degree of tolerance and self-restraint and in the circumstances of D, might have reacted in the same or in a similar way to D". Thus, the provocation partial defense requires: "1. A loss of self-control (not necessarily sudden), 2. D's loss of control must have been attributable to one or both of two specified "qualifying triggers": (i) D's fear of serious violence from V against D or another identified person and/or (ii) Things done or said (or both) which (a) constitute circumstances of an extremely grave character, and/or (b) cause D to have a justifiable sense of being seriously wronged" (Ormerod, 2011; *p*. 508).

4.3. Criminal dangerousness

Finally, we would like to add a note on the criminal dangerousness of individuals with MAOA-L. Above we have concluded that the interaction between such genotype and severe maltreatment in the early stages of life represents a vulnerability consisting of an abnormal impulsivity and a higher risk of an aggressive reaction to a triggering event. Legally, that may permit the possibility of a plea for mitigation based on the grounds of a diminished capacity for self-control and thus, a diminished responsibility. It must be stated openly that such a profile can be immediately translated into individual prognosis of criminal dangerousness.

The empirical evidence summarized above provided support for the biological etiology of the risk for antisocial behavior. This opens the door to two criminal policy orientations of radically opposite valence (Aspinwall, Brown, & Tabery, 2012; Farahany & Coleman, 2006). As we have discussed, on the one hand, this information could be used for a possible waiver or reduction of criminal responsibility to give the individual a fair consideration. However, on the other hand, it also brings up the need to control the individual's dangerousness through security measures, in order to protect the society. It will always, therefore, remain "a double-edged sword" (Aspinwall et al., 2012; Baum, 2011).

The result, given the way criminal laws are made today by politicians, is rather unpredictable. However, a realistic glance indicates that the standard of safety, protection of society, and social alarm will prevail before the rights of the individual, the defendant. Paradoxically, calling for a greater legitimacy of criminal law, recognizing culpability deficits based on the right to equality, will most likely result in, and will be consistently justifying a much more severe global criminal response; through, for example, the introduction of generic treatment measures or introduction of these facets as aggravating factors (Beecher-Monas & Garcia-Rill, 2006).

For this reason, as scientific evidence has only demonstrated effects of the interaction between MAOA-L and early maltreatment, such that it would permit invoking only a possible mitigation of the individual's criminal responsibility, the individual's dangerousness should be evaluated with equal weight. These effects drastically deny the legitimacy of further security measures or any aggravation of the criminal punishment. Given the current state of scientific knowledge, as we proposed here, lawmakers and court officials could rightfully assume diminished responsibility based on the research findings. However, given the lack of specific treatments and a number of aspects requiring further understanding, it would be difficult to justify a more severe legal response to these cases. Uncertainty and risk of mistakes should always fall on the side of the less costly solution.

This will be the panorama and the risk. New scientific studies gradually reveal more about the biosocial roots of antisocial behavior. Asked by science to take into consideration its findings, Law will always wield a double-edged sword. It cannot and does not mean, however, that science should not be allowed to advance in this field. It is only that, at the same time, science should also focus on providing effective treatment alternatives, preferably minimally invasive, affordable and respectful of human dignity. This should be, in our view, the highest priority for behavioral genetics and neuroscience, in order to be useful and enlightening to the criminal law and the society overall.

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