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LETTERS TO THE EDITOR

Presidential pardon and mentally ill offenders detained in forensic hospitals

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The presidential pardon is a Brazilian tradition enacted every Christmas season by federal decree. An inheritance from Portuguese tradition, the Imperial pardon was incorporated into the first Brazilian constitution, of 1824, and remains a presidential prerogative according to the 1988 Federal Constitution.[1] In 2008, this merciful regulation included, for the first time, mentally ill offenders detained in forensic hospitals (decree 6076/2008).[1] Since then, it has been renewed on an annual basis.

According to the Brazilian Penal Code (1940, revised 1984), when a defendant is unable to understand the illicit nature of his or her acts or is incapable to behave in accordance with his or her understanding, he or she will be found ''unimputable'' (not subject to criminal responsibility).[1,2] Defendants thus found will be pronounced not guilty by reason of insanity (NGRI) and sentenced to compulsory treatment, a type of criminal commitment known as a ''safety measure.''[1,2] The modality of treatment and its length are determined by law. An annual psychiatric risk assessment is required, as patients held under a safety measure are, by legal definition, ''dangerous.''[2] However, the new presidential decrees (issued from 2008 on) explicitly do not require a medical examination for a full pardon. These presidential decisions have been confirmed by two superior courts: the São Paulo State Court and the Superior Tribunal of Justice, in Brasilia. These peculiar decrees and upcoming sentences raise a series of questions.

First, patients in safety measure are sent to a forensic hospital for treatment, not for punishment. Thus, the logical rationale would be to follow a multidisciplinary treatment plan and discharge them only when properly treated. If this is not accomplished, some patients will be released when still under treatment, to the detriment of their best interests. Second, the forensic population is a heterogeneous one, and comprises from chronic mental patients who have committed minor offenses to psychopathic serial killers. Thus, these decisions can be a mistake from both the human rights and social security standpoints; the former because some pardoned patients are released to the ''freedom of the streets'' or removed to non-forensic psychiatric hospitals. As it is widely known, these hospitals do not have secure facilities, and their staff lacks expertise in treating forensic patients. Depending on their clinical characteristics, these patients can put the staff and other patients at risk, clearly contradicting Law 10.216/01 and basic human rights. Disregarding the risk assessment (the ''cessation of dangerousness'' examination) will place a large number of citizens at risk, since some dangerous criminals may be released only to commit new offences.[2]

Since these patients were declared NGRI, is the presidential pardon applicable? Is it ethical and legal to cease ongoing treatment abruptly? Should the safety of society and the routines of non-secure hospitals be put aside?

The authors do hope that legislators will seek guidance from mental health professionals regarding the field of mental health, and, furthermore, that future instances of the presidential pardon take into account professional opinions when dealing with mentally ill offenders.

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Disclosure

The authors report no conflicts of interest.

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HMNC1 gene polymorphism associated with postpartum depression

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Postpartum depression (PPD) is a frequent condition with major consequences for both mother and child.[1] A genetic determinant for PPD has been suggested by several reports. The first genome-wide study of PPD was recently published, and showed that the hemicentin-1 (*HMNC1*) gene had the strongest association with postpartum mood symptoms.[2] This gene encodes an extracellular protein that contains four estrogen receptor-binding sites and is involved mainly in cell migration, protein anchorage, and the formation of hemidesmosomes in the epidermis.[3]

In view of this finding, we genotyped the rs2891230 single-nucleotide polymorphism (TaqMan® SNP genotyp-

Table 1 Demographic characteristics and health status during and after pregnancy stratified by genotype, mean ± standard deviation or n (%)

	GG (n=43)	GA (n=50)	AA (n=17)
Age (years)	29.44±6.07	30.78±5.23	31.0±6.48
Higher education	22.0 (51.16)	19.0 (38)	11.0 (64.71)
Married	35.0 (81.4)	42.0 (84.0)	13.0 (76.47)
Previous pregnancies	1.53±0.99	1.68±0.73	1.52±0.60
Previous deliveries	1.33±0.64	1.48±0.61	1.41±0.49
Working out of home	30.0 (69.77)	35 (70.0)	10.0 (58.82)
EPDS score	7.93±6.2	10.32±6.75	5.94±3.06
Timing of interview (days after delivery)	58.67±11.40	59.32±12.76	57.29±8.26

ing assay, Applied Biosystems Inc, Foster City, CA, USA) of the *HMCN1* gene using a 7500 Real-Time PCR System (Applied Biosystems Inc, Foster City, CA, USA), in allelic discrimination mode. Association analysis was performed using UNPHASED software (v.3.0.14). All tests were two-tailed and the results were considered significant when $p \leqslant 0.05$. At least 10% of the samples were retyped for quality control. Sociodemographic categorical variables were analyzed by the chi-square test, and continuous variables, by analysis of variance (ANOVA).

A sample of 110 randomly selected, unrelated Brazilian women of European descent who had given birth was assessed. All subjects completed the Edinburgh Postpartum Depression Scale (EPDS) and a structured psychiatric interview (MINI PLUS 5.0), conducted by a psychiatrist, 8 weeks after delivery. The local ethics committee approved this study, and all participants signed a written informed consent form.

Following the structured psychiatric interview, 34 women (30.9%) were diagnosed with PPD. Although high, this prevalence is consistent with some previous Brazilian studies, such as those of Lobato et al.[4] and Ruschi et al.[5] No significant statistical differences in terms of sociodemographic data were observed between depressed (PPD+) and non-depressed women (PPD-). Sociodemographic data, diagnosis, and EPDS scores are shown in table 1.

The genotypic frequencies in PPD+/PPD- women were as follows: AA (0.03/0.21), GA (0.74/0.33) and GG (0.23/0.46). Genotype distribution was in Hardy-Weinberg equilibrium ($p = 0.69$). The GA genotype was associated with the presence of depressive symptoms in the postpartum period (chi-square = 15.64; $p < 0.01$; df = 2).

To the best of our knowledge, this is the first association study based on a candidate gene approach to confirm that a *HMCN1* polymorphism (rs2891230) is associated with PPD diagnosis. Heterozygosity (GA) for this SNP was associated with an increased risk of PPD. This could be an example of the phenomenon of molecular heterosis, defined as a situation in which heterozygous display a lesser or greater effect on the trait than homozygous groups. One possible explanation is an interaction with other genetic or nongenetic risk factors that causes a hidden stratification of the study population. Some limitations of our study must be considered, not least the sample size of only 110 women, but our finding confirms and provides more evidence of the importance of the *HMCN1* gene in PPD. Keeping this limitation in mind, we studied a homogeneous sample of women using a structured diagnostic interview (Mini International Neuropsychiatric Interview, MINI-PLUS). We suggest that the role of *HMCN1* in PPD should be further investigated and clarified, preferably in studies with larger samples.

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