

density lipoproteins<sup>11</sup> and seems to contribute to atherosclerosis progression by increased expression of endothelial adhesion molecules and leukocyte migration.<sup>12</sup> It is also possible that it increases the activity of plasminogen inhibitor activator.<sup>13</sup> Recently, a relationship has been suggested between endothelial dysfunction, coronary artery calcification and high C-reactive protein values in chronic consumers.<sup>14</sup> Coronary vasoconstriction, that occurs after cocaine consumption, may produce repeated injury to the arterial wall that could favor endothelial damage, thus serving as sites of platelet aggregation.<sup>6</sup> As a probable result of these pathological mechanisms, and in support of the abovementioned idea, premature coronary arteriosclerosis has been identified in autopsies in young cocaine consumers.<sup>15</sup>

Although when chest pain occurs after its consumption the percentage of acute coronary events is relatively low (6%-12%),<sup>16,17</sup> it may be worthwhile investigating the association between cocaine and chest pains in patients under 45 years old and, in particular, make health workers aware of the need to disseminate knowledge regarding this association and make consumers aware of the potential dangers of chronic consumption regarding the cardiovascular system.<sup>15,18</sup>

Guillermo Burillo-Putze,<sup>a</sup> Robert S. Hoffman,<sup>b</sup>  
and Antonio Dueñas-Laita<sup>c</sup>

<sup>a</sup>Servicio de Urgencias, Hospital Universitario de Canarias, Tenerife, Spain.

<sup>b</sup>New York City Poison Control Center, Bellevue Hospital Center, New York University Medical Center, New York, USA.

<sup>c</sup>Unidad Regional de Toxicología Clínica, Hospital Del Río Hortega, Valladolid, Spain.

## Cocaine As a Possible Emerging Cardiovascular Risk Factor

### To the editor,

In Spain the number of cocaine seizures, requests for detoxification treatment and stated consumption of this drug is increasing every year. It has been estimated that 5% of students aged between 14 and 18 years old have consumed it,<sup>1</sup> in addition to the fact that, from the cardiological standpoint, its use is worryingly associated with that of tobacco.<sup>2</sup> In the Spanish hospital emergency service cocaine represents the leading cause of acute poisoning by illegal substances.<sup>3</sup>

We have read with great interest the excellent review by Sambola et al on the effect of coronary risk factors on endothelial dysfunction and their thrombotic complications.<sup>4</sup> However, we were disappointed that cocaine consumption as a possible emerging vascular risk factor was not referred to, which in our experience probably should be added to the classic factors mentioned in the article.<sup>5,6</sup>

In addition to its acute effects at the coronary and, in general, cardiovascular levels,<sup>5,7</sup> it has been suggested that cocaine consumption increases platelet aggregation,<sup>8-10</sup> causes endothelial damage by increasing cell permeability to low-

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genetic deficiencies (antithrombin III, protein C, and protein S), catecholamine release, and stress, among others, that are involved in the pathogenesis of acute coronary syndromes.<sup>5</sup>

Our review article is basically devoted to the role of classic cardiovascular risk factors in blood thrombogenicity and, thus, although there is evidence that cocaine seems to trigger an atherothrombotic process, it has not been included as a classic risk factor, given our understanding that cocaine consumers represent a minority in society. Unfortunately, it cannot be ruled out that in the future, due to increased use, cocaine consumption may have to be tacitly included as a classic factor; however, this is not yet the case. This does not imply that any patient less than 45 years old admitted for acute coronary syndrome should not be questioned concerning consumption or those in whom there is no clear risk factor that can be associated with the presentation of a coronary event.

Antonia Sambola,<sup>a</sup> Valentín Fuster,<sup>b</sup> and Juan J. Badimon<sup>b</sup>

<sup>a</sup>Servicio de Cardiología, Hospital Universitario Vall d'Hebron, Barcelona, Spain.

<sup>b</sup>Zena and Michael A. Wiener Cardiovascular Institute, The Mount Sinai School of Medicine, New York, USA.

## Response

### To the Editor,

We would first like to thank Burillo-Putze et al for their letter. In response to their suggestions concerning the inclusion of cocaine consumption as a cardiovascular risk factor, we are in complete agreement regarding the prothrombotic role of cocaine. We are aware that several mechanisms involve cocaine in the pathogenesis of acute coronary syndromes, such as platelet hyperreactivity, probably as a consequence of the endothelial dysfunction that can be triggered by it.<sup>1-4</sup> On the other hand, in addition to cocaine consumption, the prothrombotic role of a high number of "non-classic" cardiovascular risk factors should be considered, such as homocystinemia, hyperfibrinogenemia, some

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