

Alcohol abuse in heroin addicts: An unfolding metabolic destiny

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Summary

This paper deals with the issue of alcohol-abusing heroin addicts. On the basis of clinical and epidemiological findings, a view is presented which links the two kinds of abuse along a common metabolic pathway. Some data about the former history of opiate abuse in treatment-seeking alcoholics help to indicate which heroin-related features may influence the incidence and severity of alcohol abuse in heroin addicts. Observations point to alcohol abuse as one possible pathological outcome of the opioid metabolic impairment underlying heroin addiction. When alcohol is a surrogate for heroin, social adjustment improves, but the metabolic destiny does not change, and the medical outcome is worsened to some extent by the low chances of curing a possible actual alcoholism to come. Correctly handled agonist treatments are crucial in preventing that kind of negative outcome, whereas alcohol abuse as an opioid equivalent calls for greater attention, to allow adequate assessment of the effectiveness of treatment programmes for opiate addiction.

Key Words: Heroin Addiction - Alcoholism - Metabolic
Disease - Agonist Treatment

On the heroin side: alcohol abuse by heroin addicts undergoing treatment

The available data agree in indicating that a history of alcohol abuse is quite common in addicts entering Methadone Maintenance Treatment Programmes (MMTPs), while the impact of MMTPs on pre-existing alcohol abuse is highly variable, in spite of a similar grade of effectiveness on heroin use. A sharp decrease has, in fact, been reported for some populations ^(4,5,9,24), but post-treatment cases of severe alcohol abuse are possible, especially due to the exacerbation of pre-existing abuse liability ^(7, 25). Moreover, the

possible increase in alcohol consumption during MMTPs develops along dwindling heroin use, suggesting a negative correlation between the two, at least in programmes employing lower dosages of methadone ⁽¹⁾. In other cases, alcohol abuse appears to be positively correlated with polyabuse ^(6, 8, 26-29).

In the PISA-SIA Group experience, alcohol use is common among heroin addicts at treatment entrance, whatever treatment option is applied for. Its prevalence rate is 48.8% among subjects entering a MMTP, 35.5% among those applying for a Buprenorphine Maintenance Treatment Programme (BMTP) and, surprisingly, as high as 53.5% among addicts selected to start a Naltrexone Maintenance Treatment Programme (NMTP) ⁽¹³⁾. Rates are quite similar between MMTP and NMTP, for which latter different selection criteria are employed. Generic safety criteria are the only threshold for entry into the Pisa-MMTP, whereas subjects applying for a NMPT have to go through a baseline naloxone challenge to determine naltrexone administration feasibility. Thus, alcohol use is quite frequent among addicts who are not currently using heroin on a regular basis, so suggesting that opiate addicts may attempt to abstain, at least transiently, from opiates by resorting to alcohol consumption. The lowest prevalence rate is registered among subjects accepted for BMTP, considering that a selection bias exists among physicians, who generally welcome BMTPs as an option for addicts who show partial control of their heroin use at time of baseline evaluation.

With these premises, the meaning of baseline involvement with alcohol is not that of a higher grade of severity of the opiate-related pathology, as revealed by the lack of any influence on retention in treatment ⁽¹²⁻¹⁴⁾. In comparing the features of retained subjects in a partial vs. complete agonist programme ⁽¹⁴⁾, alcohol use is greater among methadone-maintained subjects who are more severely ill as a group. Later on, when the number of retained subjects shrinks, this difference is no longer recorded. A possible explanation is that, when the average grade of addiction severity is levelled through time by the progressive dropping out of more severely addicted subjects, heavy alcohol use tends to dwindle. Alternatively, the successful outcome of treatment programmes is linked to a reduction in alcohol consumption, but this effect is unlikely when methadone dosages stay below blocking values, as in the study sample. In France, buprenorphine has been the only available agonist treatment for years: in this situation, a lower grade of addiction severity (with a duration of addiction below ten years) and alcohol dependence were among the predictors of good response to treatment ⁽²¹⁾.

Alcohol use seems to be correlated with the severity and level of activity of opiate use disorder, without adding a heterogeneous element to the clinical picture, so that the likelihood of alcohol use control is linked with the likelihood of opiate use control by therapeutic regimens.

The rising suspicion of masked heroinism

All too often the evaluation of addiction treatment programmes is exclusively founded on the maintenance of abstinence from the original substance (street opiates) in the short

term, after enrolment. When other clinical aspects are accounted for, less satisfactory pictures may result: for instance, there is a rising trend towards alcohol use among self-detoxifying and detoxified heroin addicts who undergo naltrexone treatment, suggesting that alcohol serves as a means of compensation for the loss of heroin availability^(10,18,19). Whatever heroin craving may emerge during the achievement of detoxification and enduring abstinence, addicts may succeed in providing clean urinalyses by shifting over to cross-acting substances. In the addict's natural environment, before any therapeutic setting, alcohol consumption may compensate for the lack of heroin availability (due to poverty, somatic impairment, or temporary supply shortages), so becoming a common means of self-handling in a situation of opiate craving⁽¹⁷⁾.

For heroin addicts, who have a strong motivation to "turn over a new leaf", whatever is useful in staying detached from heroin may be resorted to on a regular basis. In the case of another addictive substance, such as alcohol or cocaine, an apparent state of remission actually takes shape as a switching form of addiction. An iatrogenic way of favouring the course towards an involvement with alcohol as a surrogate consists of omitting or interrupting an effective treatment for heroin addiction. A premature removal of agonist drugs, an easy availability of naltrexone programmes as the most suitable solution for low-severity addicts, medically supervised detoxification programmes, and drug-free regimens are all examples of interventions which directly favour, or fail to counter, a switching evolution of heroin addiction towards alcoholism. Conversely, if alcohol-abusing addicts are prompted with methadone treatment, that may forestall their alcohol consumption in the short term, so indicating a rapid direct action of opioid agonism on alcohol craving in this population⁽³⁾.

A similar mechanism may limit the usefulness of agonist treatment in drinking addicts: when heroin use is the only parameter that influences therapeutic decisions, methadone dosages may stay lower, but overall improvement may be impaired by alcohol acting as a surrogate for heroin. In animal models⁽²³⁾, rats show they prefer an alcohol-methadone mixture to either alcohol or methadone alone, the latter being the least attractive to them. In particular, a higher alcohol/methadone proportion in the mixture corresponds to a higher level of appeal. This combination may reproduce a heroin-like effect, and appeal to addicts whose craving is not completely controlled.

Authors employing a wide variety of dosages were able to compare a group of subjects treated with high dosages (above 100 mg/die), with the others. While the results of heroin use showed only slight differences, higher methadone dosages were correlated with lower rates of alcohol and benzodiazepine use^(15,16). In our personal experience, we have ascertained the relationship between methadone dosage and depressant abuse in the same subject: an increase in methadone dosage was soon followed by a significant decrease in alcohol and benzodiazepine use⁽¹¹⁾. Programmes relying on low methadone dosages may therefore be spoiled by a high incidence of alcohol abuse, mirroring the incomplete control of opiate craving in the guise of alcohol-methadone coupling. As a result, it seems that methadone treatment, as long as no other feature is specified, actually favours the development of alcohol abuse⁽²⁾.

Alcohol use in a high-threshold methadone maintenance programme

As for methadone programmes, the variability of alcohol use may depend on treatment features, chiefly dose and duration.

The PISA-SIA Group MMTP provides flexible dose long-term maintenance, in which effective dosages are reached, as a general rule deriving directly from physicians' decisions, with no dose threshold, no duration barrier, and no room for rule manipulation by negotiation. Patients are given methadone at increasing dosages, on the basis of weekly clinical assessment and urinalyses, until clinical stabilization is reached (i.e. no more than one positive urinalysis for morphine in the previous two months and a DMS-IV GAF score above 60), so defining a positive outcome. Patients who fail to achieve stabilization by their first year of treatment are discharged from the programme. For those who are stabilized, any requests for dose decrease before two years of successful maintenance should be discouraged. With these premises, alcohol and cocaine use was measured by means of the Alcohol Craving Scale and the Cocaine Problem Severity Index, respectively ⁽²²⁾.

Alcohol abuse has a 37.7% frequency (20 out of 53 consecutive responders at one year); in most cases it is linked with pre-existing involvement with alcohol (primary alcoholics or dually addicted individuals). The most likely impact of methadone treatment upon alcohol use is to reduce its relevance as a clinical problem, or even to end alcohol consumption altogether. Methadone dosage is not fixed at a higher level for alcohol abusers, which may indicate indirectly that, for these subjects, detachment from heroin use is partly achieved by methadone action, and partly by alcohol use. Extending Anglin's hypothesis to all opiates (abused or therapeutic), opiate balance is self-handled by resorting to alcohol and/or opiates, in an inverse relationship. Other authors had also reported that a higher dosage is required for non-alcoholic addicts ⁽²⁰⁾.

As long as subjects abuse cocaine alone, methadone dosage is significantly higher (130 vs. 65 mg/day on average, $F 2.89$, $p = 0.04$). When alcohol and cocaine are used together, no difference is registered, which may indicate an opiate-boosting function of alcohol, automatically limiting the need for methadone coverage. When subjects abuse alcohol as well as cocaine, the need for increased methadone dosages, as observed in cocaine-only users, is neutralized so suggesting that methadone and alcohol share a cocaine-counteracting property. In this sense, Anglin's hypothesis could be extended to comprise substances, such as cocaine, which alter the opioid balance, and so induce the practice of resorting to pro-opioid factors in order to reverse their anti-opioid effect. In both cases, alcohol appears to function as an opioid surrogate, unluckily not equivalent to methadone because of its long-term toxic effects and the intrinsic addictive liability it brings with it.

The Opiate Background of Alcoholics

To acquire the advantages of an alcoholologist's point of view, we reviewed 99 consecutive files of subjects visited at Rome "La Sapienza" University's outpatient

treatment centre for alcohol-related pathology; they had all systematically undergone psychiatric evaluation based on DSM-IV criteria in conditions of sobriety. Fifteen subjects had a history of heroin addiction, but none of them had gone through any period of regular heroin use during the previous year (4 had used it at least once in the last year, 8 had not been using it for years). No differences emerged in terms of socio-anagraphical features or in chronology or quantity of alcohol use: age of first use, age of habitual involvement with alcohol consumption (i.e. daily regular use or recurrent intoxication occurring more than once a week), time interval from first use to habitual use, years of habitual use, amount of consumption at the beginning of habitual use and maximum amount ever consumed. Heroin addicts had greater psychiatric comorbidity, accounting for DSM-IV axis I psychiatric disorders, addictive disorders included, but excluding simple substance abuse ($t = -5.85$; $P < .001$). When addictive disorders alone were considered, the divergence was sharper ($t = -7.79$; $P < .001$). No differences were noted as regards the prevalence of axis II personality disorders. Psychiatric comorbid disorders were found in 64%: affective disorders were first, in order of prevalence, anxiety disorders were far less well represented, and non-affective psychoses were rare. This order of prevalence was maintained when excluding substance-induced psychiatric disorders with a possibly transient course, so ruling out a sample of undoubtedly dual diagnosis alcoholics. Age of first contact with alcohol was, predictably, lower than that of first heroin use, whereas age of onset of alcohol dependence was usually higher. In most cases, the increase in alcohol consumption took place during periods of medically supervised withdrawal of agonist therapy, or after the accomplishment of drug-free residential programmes, or spontaneously during detachment from heroin use.

Psychiatric diagnosis was classified around the bipolar spectrum, dividing the sub-sample of dual diagnosis alcoholics into bipolars and non-bipolars; no significant differences emerged. Since bipolar II was the most frequent bipolar picture, the bipolar II/non-bipolar II dichotomy was also examined: a trend ($p = 0.07$) towards a greater prevalence of bipolar II subjects among alcoholics classified as “ex-heroin addicts” was observed.

Furthermore, when looking back on the therapeutic history of former heroin addicts who later applied for treatment as alcoholics, the most likely condition is the absence of any previous specific intervention. Two out of three had never entered an agonist-based treatment programme. For those who had entered one, maximum dosages were not always in the blocking range of 80-120 mg/day or above, and were not maintained in the long term, but tapered. Duration of methadone programmes was variable.

Conclusions

It seems that alcohol addiction can be viewed as a possible negative outcome of ongoing heroin addiction. Actual alcohol addiction is not the rule among heroin addicts. Nevertheless, alcohol abuse may set in or become worse in iatrogenic conditions, such as agonist treatment omission, ineffective dose administration, opiate-free regimens,

premature medication tapering or suspension. Adopting a different viewpoint, alcoholics with a history of heroin addiction proved in most cases to have undergone no agonist treatment or to have been treated with unjustified limitations on dosage or duration. This subgroup of addicts showed a higher level of engagement in substance abuse, as testified by a higher number of addictive diseases, but did not differ from their single diagnosis alcoholic peers as regards alcohol-related parameters. Alcohol-abusing heroin addicts seem to suffer from a metabolically acquired stain, which derives from preconditioning opiate abuse, and later prompts either opiate- or alcohol-seeking behaviour in an addictive way. The accomplishment of this metabolic destiny through a shift from heroin to alcohol use is subtle, as it may be mistaken for the remission of opiate addiction, whereas it appears to be just another pathological dynamic leading to opiate use extinction. Like the so-called masked depressive syndromes, alcohol abuse in former heroin addicts can be considered a masked form of heroinism, or a sign of enduring opiate dysfunction disguised as remission. The shift from heroin to alcohol also means the transition from a highly curable disease, as heroin addiction is, to one that is hard to cure, as alcoholism is. Alcohol abuse should be taken into account when judging which agonist dosage is adequate and whether treatment outcome is satisfactory on a prognostic basis. In other words, in cases of masked relapse / alcohol abuse with morphine-free urinalyses / agonist treatment should be resorted to, as with active heroin users, since alcohol abuse is likely to be a clinical sign of unhealed, persisting opioid damage.

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