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Weekend alcoholism in youth and neurocognitive aging

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Numerous studies have shown that alcohol intake causes neuropsychological disorders that affect various brain structures. The «premature ageing» hypothesis proposes that the brain areas of alcoholics undergo deterioration similar to that observed in old age. We investigated whether alcohol abuse by young people (binge drinking) causes alterations comparable to some found in elderly people. Ninety-one people were divided into four groups: a) young people who abused alcohol; b) young people who drank alcohol in moderation; c) young people who did not drink alcohol; and d) elderly adults without any significant cognitive deterioration. All of them were assessed with a neuropsychological battery. We observed some similarities in the results obtained by young drinkers and the elderly participants, which would provide some support for the hypothesis of premature aging. The tasks that young drinkers performed worse were those related to executive functions, in which the prefrontal cortex plays an essential role. We also found differences between the two groups of young drinkers (moderate and high consumption), which leads us to believe that the amount of alcohol consumed and the pattern of consumption are factors to consider in relation to cognitive impairment.

Alcoholismo de fin de semana en jóvenes y envejecimiento neurocognitivo. Numerosos estudios han demostrado que la ingesta de alcohol provoca alteraciones neuropsicológicas que afectan a diferentes estructuras en el cerebro. La hipótesis del «envejecimiento prematuro» propone que las áreas del cerebro de alcohólicos sufren un deterioro similar al observado en la vejez. Hemos investigado si el consumo abusivo de alcohol en jóvenes ocasiona algunas alteraciones comparables a las encontradas en personas de edad avanzada. Noventa y una personas fueron divididas en cuatro grupos: a) jóvenes que abusaban del alcohol; b) jóvenes que bebían con moderación; c) jóvenes que no bebían alcohol; y d) ancianos sin deterioro cognitivo significativo, y evaluados con una batería neuropsicológica. Observamos ciertas similitudes entre los resultados obtenidos por los jóvenes bebedores y los mayores, en línea con la hipótesis del envejecimiento prematuro. Las tareas que los jóvenes bebedores realizaron peor fueron las relacionadas con funciones ejecutivas, donde la corteza prefrontal juega un papel esencial. También encontramos diferencias entre los dos grupos de jóvenes bebedores (consumo alto y moderado), lo que nos lleva a pensar que la cantidad de alcohol ingerido y el patrón de consumo son factores a tener en cuenta en relación con el deterioro cognitivo.

Alcohol, together with tobacco, is currently the most consumed psychoactive substance. This makes alcoholism one of the most important social problems in many countries, having a great impact, above all, on the younger population. Abusive alcohol consumption is starting at earlier ages and this implies family, social, and work-related difficulties. Currently, the *binge drinking* pattern is very frequent on the weekend, above all the famous «*botellón*» where alcohol brings young people together in public city spaces (Gómez-Fraguela, Fernández, Romero, & Luengo, 2008; Salamó, Gras, & Font-Mayolas, 2010).

Abusive alcohol consumption provokes neuropsychological alterations that can affect different individual capacities; in fact, around 75% of alcoholics show significant neuropsychological dysfunctions (Corral-Varela & Cadaveira, 2002; Knight & Longmore, 1994; Sher, Grekin, & Williams, 2005). The neuropsychological studies show that the most frequently affected processes are those related to executive and attention functions, visuospatial abilities, psycho-motor speed, and stability and balance (Fama, Pfefferbaum, & Sullivan, 2004; Noel et al., 2001; Ratti, Bo, Giardini, & Soragna, 2002; Rosselló, Munar, Justo, & Arias, 1998; Sullivan, Rosenbloom, & Pfefferbaum, 2000). On the contrary, other functions remain fairly preserved, particularly those related to linguistic abilities and sensoperceptual and primary motor functions (Oscar-Berman, Shagrin, Evert, & Epstein, 1997; Sullivan et al., 2000). Regarding memory, there is a lot of variability in the results, although it appears that the primary deficits affect episodic declarative and working memory, and not as much on semantic and procedural memory (Landa, Fernández-Montalvo, & Tirapu, 2004).

For a long time, this heterogeneous pattern of results has led to the proposal of several theories to explain the cognitive deficits associated with alcoholism: a) hypothesis of the differential sensitivity of the right hemisphere to the poisoning effects of alcohol,

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which indicates a greater vulnerability of those neuropsychological functions managed by the right hemisphere (visuoperceptual and visuoconstructive tasks) opposed to the greater integrity of those depending on the left hemisphere (linguistics tasks) (Ellenberg, Rosenbaum, Goldman, & Whitman, 1980; Evert & Oscar-Berman, 2001); b) hypothesis of diffuse brain damage, which proposes that alcohol progressively damages the cortico-subcortical structures in relation to the quantity and time of alcohol consumption (Beatty, Hames, Blanco, Nixon, & Tivis, 1996; Tivis & Parsons, 1995); and c) the hypothesis of frontal damage, based on the similarity in the neurocognitive deficit profile between patients with prefrontal damage and chronic alcoholics in tasks that imply conceptualization and executive capacities. Indeed, alcoholics have difficulties with attention and working memory, in the formation and use of abstract concepts, in the generation of strategies and flexible thinking when solving cognitive problems, etc., in other words, aspects that largely describe the executive functions (Ihara, Berrios, & London, 2000).

Ageing is often accompanied by problems of learning and memory, many of which resemble the deficit associated with hippocampal damage. In fact, it seems memory is one of the earliest mental functions to be affected in the ageing process (Ska & Joanette, 2006; Weaver, Maruff, Collie, & Masters, 2006). Some results suggest that the hippocampus undergoes structural and biochemical changes during normal ageing and that these changes may be an important component in cognitive deterioration associated with age (Driscoll et al., 2003). Cognitive deterioration may result from the consumption of substances such as alcohol or the ageing process itself. In this study, we wish to focus on cognitive deterioration associated with adolescent consumption of alcohol and its possible similarities with the normal ageing process. We have based it on a hypothesis, formulated many years ago, that relates cognitive deficit derived from the toxic effects of alcohol with deficits observed in normal ageing: the *premature ageing hypothesis* (Ryan, 1982; Ryan & Butters, 1984). This proposes that, as a consequence of alcohol consumption, areas of the brain undergo deterioration similar to that found in old age, and that chronic alcoholics with serious cognitive deficits resemble patients affected by dementia with generalized brain deterioration (Graff-Radford, Heaton, Earnest, & Rudikoff, 1982; Nilsson, Backman, & Karlsson, 1989). Therefore, the aim of the study is to determine, by means of a neuropsychological battery, whether the neurotoxic effects of alcohol produce some sort of cognitive deficit in young people and whether there are similarities between this deficit and that observed in normal ageing process in the elderly.

Method

Participants and procedure

To select the sample, young people completed a questionnaire with items about their alcohol consumption, and the elderly, about their habits and customs. 91 people (53♀ - 36♂) that meet the criteria previously established related to alcohol intake, age or cognitive status. They get into four groups: a) BD group, youth who consumed more than 6 (women) or 8 (men) units of alcohol (AUs) during a single session of two/three hours (n= 21, 8♂ and 13♀, mean age 18.97±1.19; mean AUs ♂= 11.68±6.12 and ♀= 7.62±2.29); ALM group, young people who drink less than 6 (women) or 8 (men) AUs (n= 24, 9♂ and 13♀, mean age 19.01±1.39; mean AUs ♂= 2.15±1.08 and ♀= 2.62±1.24); CTR group, young people that didn't consume alcohol (n= 20, 8♂ and 12♀, mean age 18.76±1.67); and AGN group, people 65 years older without significant cognitive decline (*Mini Mental State Examination* ≥ 25) and that didn't consume alcohol (n= 26, 11♂ and 15♀, mean age 69.35±4.82). We excluded subjects who consumed other drugs (except tobacco) or with a history of psychological, psychiatric or neurological disorders. The subjects from the ANC group were abstinent, and at least had no consumed alcohol in the last five years. We have also excluded subjects who had seizures, fainting or, in the days before the evaluation, extraordinary situations. All of the subjects were evaluated in individual sessions lasting approximately 50-60 minutes that were held on Tuesday, Wednesday and Thursday.

Instruments

TAVEC: Based in the California Verbal Learning Test (CVLT) (Delis, Kramer, Kaplan, & Ober, 1987). It evaluates verbal learning in tasks involving immediate and delayed memory and recognition.

Tower of Hanoi: The objective is to shift five discs, moving one by one each time, from position A to C and so as to maintain the same pyramid and without placing a larger disk above a smaller disk. The time and the number of movements used in the resolution were evaluated and utilized to obtain direct score (DS) according to the next formula:

$$\mathbf{DS}= 100 - \{[(M-27) \times 0.5] + [(T-60) \times 0.09]\}$$

M: Number of movements
T: Time in seconds
27: Minimum number of movements necessary
60: Seconds estimated to solve the problem in youth (120 in ANC group)

People obtained 0 points if they didn't solve the problem in 10 minutes or if they didn't built a tower of four disks in 5 minutes.

Stroop color-word test: Designed to evaluate the capacity to avoid automatically generated responses thanks to an inhibition process. It is considered a general measure of cognitive flexibility and control or executive functioning.

Digits and Corsi Blocks of the Wechsler Memory Scale: These tests allow for the evaluation of immediate memory with verbal or visuospatial material in a forward and backward ways.

BVRT (Benton's Visual Retention Test): In this activity, the subject should reproduce ten complex pictures by memory after observing them for 10 seconds.

The subjects were also evaluated through the *STAI* to determine the extent to which the context itself in which the evaluation is held could be influencing the subjects' execution of different tasks.

Data analysis

We considered as independent variable both the pattern of consumption and aging to look for differences and similarities between the effects of alcohol and aging. Then, and to determine which tasks appear, we performed an ANOVA for each test applied and post-hoc analysis with the Student-Newman-Keuls test. The minimum statistical significance were established in $p<0.05$. All analysis were performed using the SPSS 15.0 for Windows program.

Results

We found significant differences in verbal memory tasks, both in immediate (F= 17.145; p<0.001), and short- (F= 14.564; p<0.001) and long-term recall (F= 11.917; p<0.001). The post hoc analysis didn't show significant differences in the performances of the three groups of youth; however, they showed significant differences with respect to the group of elderly people (p<0.01 and p<0.05), though neither the BD group or the ALM group performed as well as the control group (Fig. 1). We found significant differences in three indices of mnesic accuracy: perseverations (F= 4.376; p<0.005), intrusions (F= 4.105; p<0.05) and false positives (F= 10.941; p<0.001). There were more intrusions and false positives in the group of elderly people than in the three groups of young people (p<0.01 and p<0.05). Nevertheless, it was the groups that consumed alcohol that committed the most perseverations, not only more than the control group but more even than the AGN group (p<0.01 and p<0.05) (Fig 2).

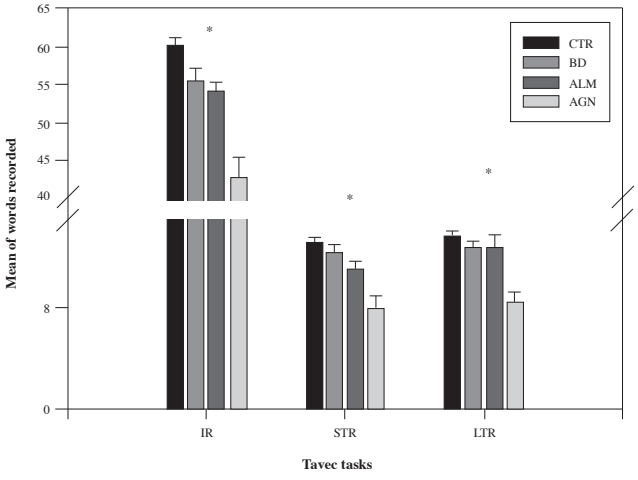

Figure 1. *Verbal memory performance in Tavec tasks (RI: immediate recall, STR: sort-term recall, LTR: long-term recall. See explanation about statistical significance in the text)*

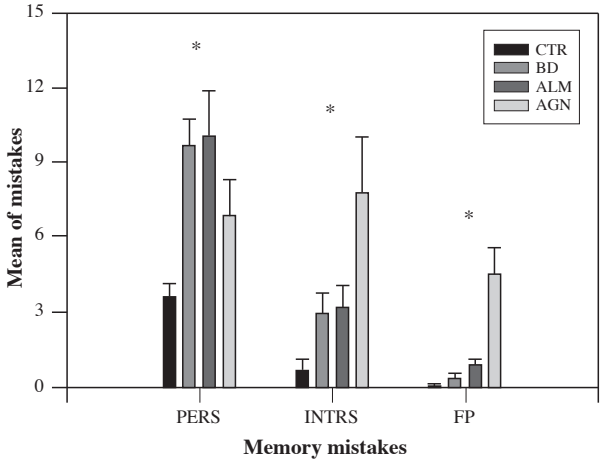

Figure 2. *Mistakes in verbal memory performance (PERS: perseverations, INTRS: intrusions, FP: false positives. See explanation about statistical significance in the text)*

We also found significant differences in the Digit and Corsi tests (DIG: F= 15.237; p<0.001; CRS: F= 12.768; p<0.001). In the digits forward tasks, a posteriori analysis showed that results obtained by the group of elderly were similar to those obtained by young people who consumed alcohol and the results of these three groups were significantly lower than those of the control group, both in digit (p<0.01 and p<0.05) and Corsi tests (p<0.01 and p<0.05) (Fig 3). The AGN group's total score was lower than those of BD and ALM groups (p<0.01 and p<0.05). Accordingly, the performance of these groups was worse than control group, due to the AGN group more difficult to recall words or positions in the backward task.

We also detect notable differences among groups in the three Stroop tasks (STRP-W: F= 6.118; p<0.001; STRP-C: F= 13.515; p<0.001 and STRP-WC: F= 12.934; p<0.001). In word reading, the control group results were significantly higher than those of the BD, ALM and AGN groups (p<0.01 and p<0.05), which had no appreciable differences between them. Something similar occurred with the color naming task and the word/color task, but in these tasks the group of elderly people performed less well than the groups of young people who consumed alcohol (p<0.01 and p<0.05). With these data, the ANG group exhibited a greater interference level (negatives scores) than young groups (F= 10.310; p<0.001) (Fig. 4). Significant differences were also found in the Tower of Hanoi test (F= 9.224; p<0.001); scores obtained by the elderly people were considerably lower than the young people's scores, without differences among drinkers and non-drinkers. Performances in the word/color task and the Tower of Hanoi test showed a similar profile which demonstrated that both the elderly people and the juvenile drinkers performed worse than the control group. Finally, in the Benton test (TRVB: F= 33.927; p<0.001), no differences were observed between the three groups of young people whose results were significantly higher than those of the AGN group (P<0.05) (Fig. 5).

Analysis of data from the STAI revealed that all the subjects showed similar anxiety levels at the moment of assessment. However, when anxiety was considered as a more stable feature of character, significant differences were found (F= 5.354; p<0.005); the control group showed a lower anxiety level than the AGN group and young people who consumed alcohol (P<0.05), with no differences between them.

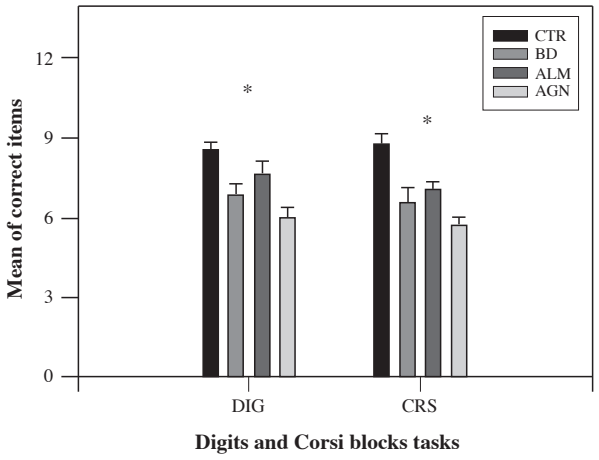

Figure 3. *Digits and Corsi blocks performance (See explanation about statistical significance in the text)*

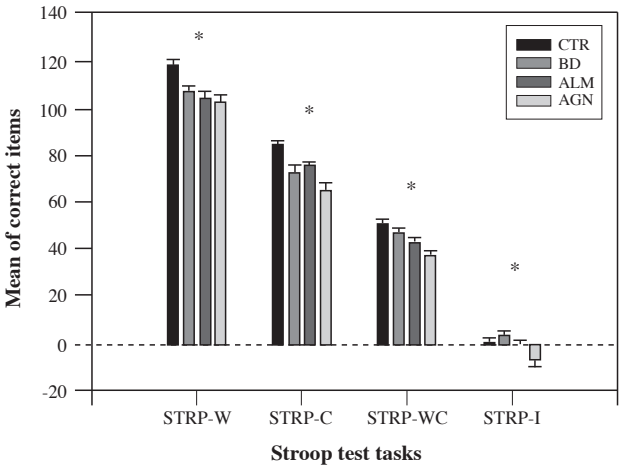

Figure 4. *Stroop test scores (STRP-W: Word reading, STRP-C: Color naming, STRP-WC: word-color task, STRP-I: Interference. See explanation about statistical significance in the text)*

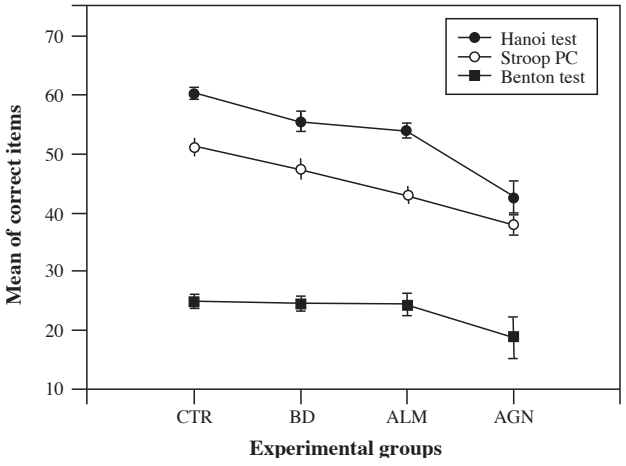

Figure 5. *Groups' performance in Hanoi, Stroop and Benton tests (See explanation about statistical significance in the text)*

In summary, the performance of the AGN, ALM and BD groups in the neuropsychological tests was generally lower than that of the control group. Moreover, in some of these tests, there was no difference in performance between students who consumed alcohol and the group of elderly people. Nevertheless, although the confidence level established was not reached in some of the differences, there does seem to be a profile indicating that the performance of young drinkers is influenced by the intensity of alcohol consumption as it was observed that BD subjects performed worse than the more moderate drinkers.

Discussion

Our results show that there are certain similarities between the performances of young people who consume alcohol (BD and ALM) and the group of elderly adults, especially the BD group. The AGN group scored significantly lower in almost all the tests, compared to the control group. It should be noted that there are about 50 years between youth groups and the elderly group (this group generally performed less well in the tests because of the inevitable diminution in cognitive performance with age). Similarities between this group and that of young people who drank alcohol were most apparent in tasks that involve executive functions. Our results coincide with other studies showing similarities between alcoholics and the elderly, as demonstrated by radiological, electrophysiological and neuropsychological indicators and by regional blood flow distribution in the brain (Ciesielski, Waldorf, & Jung, 1995; Noonberg, Goldstein, & Page, 1985; Ryan & Butters, 1984). It has been found that the mental age of alcoholic patients is approximately 7 years higher than that of normal subjects of the same chronological age, revealing a faster ageing process in alcoholic people (Holden, McLaughlin, Reilly, & Overall, 1988).

Several research argues that, both in the cognitive and neuropsychological sense, alcoholics become old before their time (Holden et al., 1988; Kramer, Blusewicz, & Preston, 1989; Noonberg et al., 1985).This type of data has raised speculation that excessive alcohol consumption causes premature ageing of the brain, which leads to the characteristic structural and functional changes seen in detoxified alcoholics (Graff-Radford et al., 1982; Noonberg et al., 1985; Ryan & Butters, 1984). The results of our study are in line with the hypothesis of that, a kind of premature ageing of the neuropsychological functions, and possibly of the brain, is caused by alcoholism.

However, does it mean that alcohol and aging share mechanisms to produce cognitive decline? Several investigations have provided empirical supports for the idea that there are some processes that are impaired by both aging and alcoholism. However, the findings from large standardized batteries have shown that some of the effects of aging and alcoholism are similar, although the effects could be mediated via dysfunction of different mechanisms (Glass, Park, & Zucker, 1999). The results of this research lend certain support to the hypothesis of premature ageing, since similarities in performance were detected between the groups of young people who drank alcohol and the elderly group, however, we also found that the functions most affected in the drinkers were the executive functions, which is in line with the hypothesis of the frontal lobe (Oscar-Berman & Marinkovic, 2003), a hypothesis that has a considerable empirical support. This hypothesis proposes that executive processes appear to be interrupted by alcoholism. Chronic consumption of alcohol seems to be related to a severe deficit in executive function that persists even after an extended period of abstinence. These data support the idea that cognitive deficit in sober alcoholics who have recently been detoxified is due to dysfunction of the frontal lobe (George, Potts, Kothman, Martin, & Mukundan, 2004; Noel et al., 2001).

Different lines of research suggest that abnormalities in the frontal lobe are a prominent characteristic of the alcoholic brain. This has been demonstrated both by weaker performances of neuropsychological tasks related to the functioning of this area, and by the reduced volume of this structure as seen with neuroimaging techniques (Kril, Halliday, Svoboda, & Cartwright, 1997; Paul et al., 2008). Several studies have shown that the frontal lobe structures are particularly susceptible to alcohol-induced damage (Chao, Meyerhoff, Cardenas, Rothlind, & Weiner, 2003; Kokavec & Crowe, 1999; Noel et al., 2001) and that the reduction in volume of this area is largely due to a loss of white matter (McQueeny et al., 2009). Moreover, the prefrontal cortex is the last brain region to mature, making it much more vulnerable to the toxic effects of alcohol and other drugs (Squeglia, Jacobus, & Tapert, 2009).

In this study we have seen that groups that consumed alcohol performed worse than control group in tasks related to executive functions, such as cognitive control, working memory, planning and attention. For example, in the TAVEC test, alcoholic groups showed a significantly higher number of perseverations than the CTR group, which denoted low cognitive control. A large number of perseverations are usually associated with damage to the structures making up the prefrontal system (Chirivella, Ferri, Villodre, & Noé, 2003; Davis, Price, Kaplan, & Libon, 2002). These kinds of errors on the recall accuracy are very common in the elderly too (De Beni & Palladino, 2004). One of the factors to be considered in the TAVEC test is that there are no signs of memory deterioration; nevertheless, at the beginning of the test, the BD group obtained fewer words in the first recall trial (as did the group of elderly people) than the ALM or CTR groups, although they recovered to equal their peers later on. A significantly greater increase in the number of words recalled between the first and second trial (or between the two first trials and the third) than between the last trials usually indicates difficulty in *«entering»* a new task, something frequently found in people who are anxious, who present cognitive rigidity associated with certain frontal lesions (Chirivella et al., 2003), or in aging (Traykov et al., 2007; Wecker, Kramer, Hallam, & Delis, 2005).

Within the prefrontal cortex, the *dorsolateral circuit* is associated with cognitive skills, such as working memory, selective attention, concept formation and cognitive flexibility; while the *ventromedial circuit* is associated with the processing of emotional signals that guide our decision-making towards objectives based on social and ethical judgement (Bechara, Damasio, & Damasio, 2000). Alcohol intake doesn't affect equally these two functional circuits in the prefrontal cortex. In the neuropsychological field, Scaife and Duka (2009) found that functions linked to dorsolateral prefrontal cortex were more impaired in young social drinkers with binge drinking pattern than in control subjects, whereas functions linked to orbitofrontal cortex were not impaired. Something similar was observed in aging, where MacPherson, Phillips and Della Sala (2002) found age-related differences in performance on all tasks dependent on dorsolateral prefrontal dysfunction. In contrast, they didn't found age-related differences on the tasks dependent on ventromedial prefrontal dysfunction. Those results are not definitive since other authors found, with more sensitive tasks, age-related differences on the ventromedial prefrontal cortex functioning (Baena, Allen, Kaut, & Hall, 2010) in patients who showed deficits in emotional/cognitive integration as well as in executive function.

Finally, most studies on alcohol consumption are related to chronic alcoholism rather than sporadic consumption pattern, which is what this study has addressed. We found that exist neuropsychological alterations derived from this pattern of consumption, in line with other investigations (Stephens & Duka, 2008). We found too that the performance of the BD group was poorer than that of the ALM group in most of the neuropsychological tests. That is because, with respect to cognitive damage, the quantity of alcohol consumed and the patterns of consumption are factors to be taken into consideration.

According to our data, we can conclude that a pattern of irregular alcohol consumption is enough to provoke some degree of cognitive deficit and that some neuropsychological effects of the binge drinking pattern in youth are similar those observed in normal ageing. We can't state if alcoholism produces a cognitive aging in the strict sense of the word. Moreover, the results should be interpreted with caution since issues such as gender differences or genetic predisposition may bring confusion to them (Cadaveira, 2009). Both in alcoholism and in aging, task depending on prefrontal cortex are earlier affected although the mechanisms of deterioration are not always the same in both processes. Anyway, this prefrontal damage could be a field of study for the design of intervention strategies both in alcoholic addiction and in the age-related cognitive deficits.

References

Baena, E., Allen, P.A., Kaut, K.P., & Hall, R.J. (2010). On age differences in prefrontal function: The importance of emotional/cognitive integration. *Neuropsychologia, 48,* 319-333.

Beatty, W.W., Hames, K.A., Blanco, C.R., Nixon, S.J., & Tivis, L.J. (1996). Visuospatial perception, construction and memory in alcoholism. *Journal of Studies on Alcohol, 57*, 136-143.

Bechara, A., Damasio, H., & Damasio, A.R. (2000). Emotion, decision-making and the Orbitofrontal cortex. *Cerebral Cortex, 10,* 295-307.

Chao, L.L., Meyerhoff, D.J., Cardenas, V.A., Rothlind, J.C., & Weiner, M.W. (2003). Abnormal CNV in chronic heavy drinkers. *Clinical Neurophysiology, 114,* 2081-2095.

Chirivella, J., Ferri, J., Villodre, R., & Noé, E. (2003). Test de aprendizaje verbal complutense frente a escala de memoria Wechsler-revisada. *Neurología, 18,* 132-138.

Ciesielski, K.T., Waldorf, A.V., & Jung, R.E. (1995). Anterior brain deficits in chronic alcoholism. Cause or effect? *Journal of Nervous and Mental Disease, 183,* 756-761.

Cadaveira, F. (2009). Alcohol y cerebro adolescente. *Adicciones, 21,* 9-14.

Corral-Varela, M., & Cadaveira, F. (2002). Aspectos neuropsicológicos de la dependencia del alcohol: naturaleza y reversibilidad del daño cerebral. *Revista de Neurología, 35,* 682-687.

De Beni, R., & Palladino, P. (2004). Decline in working memory updating through ageing: Intrusion error analyses. *Memory, 12,* 75-89.

Davis, K.L., Price, C.C., Kaplan, E., & Libon D.J. (2002). Error analysis of the nineword California Verbal Learning Test (CVLT-9) among older adults with and without dementia. *Clinical Neuropsychology, 16,* 81-89.

Delis, D.C., Kramer, J.H., Kaplan, E., & Ober, B.A. (1987). *California Verbal Learning Test*. San Antonio, The Psychological Corporation.

Driscoll, I., Hamilton, D.A., Petropoulos, H., Yeo, R.A., Brooks, W.M., Baumgartner, R.N., & Sutherland, R.J. (2003). The aging hippocampus: Cognitive, biochemical and structural findings. *Cerebral Cortex, 13,* 1344-1351.

Ellenberg, L., Rosenbaum, G., Goldman, M.S., & Whitman, R.D. (1980). Recoverability of psychological functioning following alcohol abuse: Lateralization effects. *Journal of Consulting and Clinical Psychology, 48*, 503-510.

Evert, D.L., & Oscar-Berman, M. (2001). Selective attentional processing and the right hemisphere: Effects of aging and alcoholism. *Neuropsychology, 15*, 452-61.

Fama, R., Pfefferbaum, A., & Sullivan, E.V. (2004). Perceptual learning in detoxified alcoholic men: Contribution from explicit memory, executive function and age. *Alcoholism: Clinical and Experimental Research, 28*, 1657-1665.

George, M.R., Potts, G., Kothman, D., Martin, L., & Mukundan, C.R. (2004). Frontal deficits in alcoholism: An ERP study. *Brain and Cognition, 54,* 245-257.

Glass, J.M., Park, D.C., & Zucker, R.A. (1999). Alcoholism, aging and cognition: A review of evidence for shared or independent impairments. *Aging Neuropsychology and Cognition, 6,* 157-178.

Gómez-Fraguela, J.A., Fernández, N., Romero, E., & Luengo, A. (2008). El botellón y el consumo de alcohol y otras drogas en la juventud. *Psicothema, 20,* 211-217.

Graff-Radford, N.R., Heaton, R.K., Earnest, M.P., & Rudikoff, J.C. (1982). Brain atrophy and neuropsychological impairment in young alcoholics. *Journal of Studies on Alcohol, 43,* 859-868.

Holden, K.L., McLaughlin, E.J., Reilly, E.L., & Overall, J.E. (1988). Accelerated mental aging in alcoholic patients. *Journal of Clinical Psychology, 44,* 286-292.

Ihara, H., Berrios, G.E., & London, M. (2000). Group and case study of the dysexecutive syndrome in alcoholism without amnesia. *J Neurol Neurosurg Psychiatry, 68*, 731-737.

Knight, R.G., & Longmore, B.E. (1994). *Clinical Neuropsychology of Alcoholism*. Hove: Lawrence Erlbaum Associates, Ltd.

Kokavec, A., & Crowe, S.F. (1999). A comparison of cognitive performance in binge versus regular chronic alcohol misusers. *Alcohol and Alcoholism, 34,* 601-608.

Kramer, J.H., Blusewicz, M.J., & Preston, K.A. (1989). The premature aging hypothesis: Old before its time? *Journal of Consulting and Clinical Psychology, 57,* 257-262.

Kril, J.J., Halliday, G.M., Svoboda, M.D., & Cartwright, H. (1997). The cerebral cortex is damaged in chronic alcoholics. *Neuroscience, 79,* 983-998.

Landa, N., Fernández-Montalvo, J., & Tirapu, J. (2004). Alteraciones neuropsicológicas en el alcoholismo: una revisión sobre la afectación de la memoria y las funciones ejecutivas. *Adicciones, 16,* 41-52.

MacPherson, S.E., Phillips, L.H., & Della Sala S. (2002). Age, executive function and social decision making: A dorsolateral prefrontal theory of cognitive aging. *Psychology and Aging, 17,* 598-609.

McQueeny, T., Schweinsburg, B., Schweinsburg, A., Jacobus, J., Bava, S., Frank, L., & Tapert, S. (2009). Altered white matter integrity in adolescent binge drinkers. *Alcoholism: Clinical and Experimental Research*, *33,* 1278-1285.

Nilsson, L.G., Backman, L., & Karlsson, T. (1989). Priming and cued recall in elderly, alcohol intoxicated and sleep deprived subjects: A case of functionally similar memory deficits. *Psychological Medicine*, *19*, 423-433.

Noel, X., Van der Linden, M., Schmidt, N., Sferrazza, R., Hanak, C., Le Bon, O., De Mol, J., Kornreich, C., Pelc, I., & Verbanck, P. (2001). Supervisory attentional system in nonamnesic alcoholic men. *Archives of General Psychiatry, 58,* 1152-1158.

Noonberg, A., Goldstein, G., & Page, H.A. (1985). Premature aging in male alcoholics: «Accelerated aging» or «increased vulnerability»? *Alcoholism: Clinical and Experimental Research, 9,* 334-338.

Oscar-Berman, M., & Marinkovic, K. (2003). Alcoholism and the brain: An overview. *Alcohol Research & Health, 27,* 125-133.

Oscar-Berman, M., Shagrin, B., Evert, D.L., & Epstein, C. (1997). Impairments of brain and behavior. The neurological effects of alcohol. *Alcohol Health and Research World*, *21*, 65-75.

Paul, C., Au, R., Fredman, L., Massaro, J., Seshadri, S., DeCarli, C., & Wolf, P. (2008). Association of alcohol consumption with brain volume in the Framingham Study. *Archives of Neurology, 65,* 1363-1367.

Ratti, M.T., Bo, P., Giardini, A., & Soragna, D., (2002). Chronic alcoholism and the frontal lobe: Which executive functions are impaired? *Acta Neurologica Scandinavica, 105*, 276-281.

Rosselló, J., Munar, E., Justo, S., & Arias, R. (1998). Efectos del alcohol sobre la atención dividida y la precisión del cambio atencional. *Psicothema, 10,* 65-73.

Ryan, C. (1982). Alcoholism and premature aging: A neuropsychological perspective: *Alcoholism: Clinical and Experimental Research*, *6,* 22-30.

Ryan, C., & Butters, N. (1984). Alcohol consumption and premature aging. A critical review. *Recent Developments in Alcoholism, 2,* 223-250.

Salamó, A., Gras, E., & Font-Mayolas, S. (2010). Patrones de consumo de alcohol en la adolescencia. *Psicothema, 22,* 189-195.

Scaife, J.C., & Duka, T. (2009). Behavioural measures of frontal lobe function in a population of young social drinkers with binge drinking pattern. *Pharmacology, Biochemistry and Behavior, 93,* 354-362.

Sher, K.J., Grekin, E.R., & Williams, N.A. (2005). The development of alcohol use disorders. *Annual Review Of Clinical Psychology, 1,* 493-523.

Ska, B., & Joanette, Y. (2006). Normal aging and cognition. *Medicine Sciences, 22,* 284-287.

Squeglia, L.M., Jacobus, J., & Tapert, S.F. (2009). The influence of substance use on adolescent brain development. *Clinical EEG Neuroscience, 40,* 31-38.

Stephens, D.N., & Duka, T. (2008). Cognitive and emotional consequences of binge drinking: Role of amygdala and prefrontal cortex. *Philosophical Transactions of the Royal Society B, 363*, 3169-3179.

Sullivan, E.V., Rosenbloom, M.J., & Pfefferbaum, A. (2000). Pattern of motor and cognitive deficits in detoxified alcoholic men. *Alcoholism: Clinical and Experimental Research*, *24*, 611-621.

Tivis, L.J., & Parsons, O.A. (1995). An investigation of verbal spatial functioning in chronic alcoholics. *Assessment, 2,* 285-292.

Traykov, L., Raoux, N., Latour, F., Gallo, L., Hanon, O., Baudic, S., Bayle, C., Wenisch, E., Remy, P., & Rigaud, A.S. (2007). Executive functions deficit in mild cognitive impairment. *Cognitive and Behavioral Neurology, 20,* 219-224.

Weaver, C.J., Maruff, P., Collie, A., & Masters, C. (2006). Mild memory impairment in healthy older adults is distinct from normal aging. *Brain and Cogntion, 60,* 146-155.

Wecker, N.S., Kramer, J.H., Hallam, B.J., & Delis, D.C. (2005). Mental flexibility: Age effects on switching. *Neuropsychology, 19,* 345-352.