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Research Article

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The Protective Effect of N-Acetylcysteine on FAS Gene Expression Level in the Ovaries of Rats Treated with Acrylamide

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Abstract

Background and aim: N-acetylcysteine is an antioxidant drug that is used to treat many disorders, including the treatment of poisoning. This study was conducted with the aim of investigating the protective effect of different doses of N-acetylcysteine on the change in FAS gene expression level in the ovarian tissue of rats treated with acrylamide as a toxic compound.
Materials and Methods: 36 adult female Wistar rats were randomly divided into 6 equal groups. The dose of acrylamide was 50 mg/kg and the doses of N-acetylcysteine were 10, 20 and 40 mg/kg. The grouping of animals included the control group, acrylamide, N-acetylcysteine and a combination of acrylamide and N-acetylcysteine. 28 days later, the ovaries of the animals were removed and FAS mRNA expression level was measured by real-time PCR method.
Results: Acrylamide alone increased the FAS expression level compared to the control group ($p<0.05$). On the other hand, N-acetylcysteine at the maximum dose (40 mg/kg) did not show a significant difference in FAS expression level compared to the control group ($p>0.05$).
Conclusion: Administration of acrylamide in rats increases FAS expression level in ovarian tissue and induces apoptosis. On the other hand, the administration of N-acetylcysteine at the maximum dose (40 mg/kg) improves FAS expression level with antioxidant effects and inhibits induced apoptosis in ovarian tissue.

Keywords: *N-acetylcysteine, Acrylamide, Ovary, FAS gene, Rat*

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Introduction

Food safety and its health is one of the major problems of today's societies. In addition to providing all the nutrients, food must be free from any contamination that can lead to disease in humans and animals. It has been proven that cooking food at high temperatures and processed food may contain toxic compounds that are considered as health risk factors (Pogurschi *et al.,* 2021). Acrylamide or 2-propanamide with the chemical formula C_3H_5NO is an organic compound that is mainly used in industry. However, studies have shown that acrylamide is produced during the cooking process of food by the Maillard reaction due to the reaction of free amino acids, mainly asparagine, with carbohydrates in food. The International Agency for Research on Cancer has classified acrylamide as a human carcinogen because it is likely to cause DNA damage and gene mutations (Kumar *et al.,* 2018). In the body, acrylamide combines with glutathione non-enzymatically or by glutathione-S-transferases through an epoxidation reaction mediated by cytochrome P-450 CYP2E1 and produces glycidamide, which is the main metabolite of acrylamide. Both compounds can bind to and damage hemoglobin, albumins, proteins, and DNA. However, glycidamide is more reactive and genotoxic than acrylamide (Nowak *et al.,* 2020). It has been shown that acrylamide can increase the content of reactive oxygen species (ROS). Supraphysiological accumulation of ROS can lead to an increase in the level of inflammatory factors, an increase in lipid peroxidation, and as a result, induction of apoptosis in different tissues (Song *et al.,* 2021). Previous studies show that acrylamide causes ovarian dysfunction by disrupting the secretion of steroid hormones and increasing apoptosis (Aldawood *et al.,* 2020). FAS protein is a member of the death receptor family that is involved in various forms of physiological and pathological cell death. FAS receptor activation by FAS ligand (FASL) triggers a complex cascade of intracellular events that ultimately leads to caspase 8 activation and apoptosis. FAS-mediated apoptosis is an essential mechanism for maintaining normal tissue homeostasis, and disruption of this death pathway is associated with numerous diseases and inflammation (Wang *et al.,* 2008).

N-acetylcysteine is a precursor of cellular cysteine and glutathione and is widely used to treat acetaminophen overdose. Recently, this drug is also used as a mucolytic drug in respiratory diseases. The main role of N-acetylcysteine is related to its antioxidant and anti-inflammatory activity, which helps to maintain the cellular redox (oxidation-reduction) balance. For this reason, the therapeutic potential of N-acetylcysteine is related to diseases associated with oxidative stress (Aldini *et al.,* 2018). However, the mechanisms by which N-acetylcysteine exerts its antioxidant properties have not yet been fully elucidated (Ezerina *et al.,* 2018). N-acetylcysteine not only has favorable and protective effects on cell activity, but it is also a safe drug with high bioavailability and cheap, that's why it is used in studies based on oxidative stress (Aldini *et al.,* 2018). The anti-ROS activity of N-acetylcysteine is attributed to its ability to react with oxygen ions. Studies show that N-acetylcysteine protects against oxidative stress-induced by cell death and apoptosis (Lin *et al.,* 2020). Therefore, this study was designed with the aim of investigating the protective effect of different doses of N-acetylcysteine on the change in FAS gene expression level in the ovarian tissue of rats treated with acrylamide.

Materials and Methods

Animals and study protocol

In this study, 36 adult, female Wistar rats were purchased from the animal house of Islamic Azad University, Kazerun branch and kept in the same place. In order to prevent stress in the animals, before starting the study, all animals were kept for 2 weeks under standard temperature conditions of 22 ± 2 °C, 12 hours light/dark, 75% humidity and kept in polycarbonate cages. These conditions were maintained until the end of the study, and all animals had free access to water and sufficient food during this period. The ethics protocol for working with laboratory animals in this study was approved by the ethics committee of Islamic Azad University of Shiraz branch (ethics code: IR.IAU.SHIRAZ.16330525651006).

Animals were randomly grouped into 6 equal groups. The control group did not receive drug treatment. AA group received 50 mg/kg acrylamide (Merck, Germany) orally. Animals in the NAC group received 40 mg/kg N-acetylcysteine (Merck, Germany) as an intraperitoneal injection. Animals of groups AA+NAC1, AA+NAC2 and AA+NAC3 received 10, 20 and 40 mg/kg of N-acetylcysteine,

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respectively by intraperitoneal injection at 9 am. and at 5 pm, they received 50 mg/kg of acrylamide orally. The dose selection of acrylamide and N-acetylcysteine was based on previous studies (Camacho *et al.,* 2012; Chiew *et al.,* 2016). The duration of the study was defined as 28 days, and at the end, all animals were anesthetized with ether (Merck, Germany) and the right and left ovaries were removed from the abdominal area. Evaluation of FAS gene expression level in rat ovaries was done by real-time PCR method.

Statistical analysis

Statistical analysis of the data of this study was done using SPSS software version 20 (SPSS Inc., Chicago, IL). The normal distribution of the data was confirmed by the Kolmogorov-Smirnov test, and then one way ANOVA and LSD post hoc test were used to determine the significance of the data between the control group and other groups at the $P<0.05$ level. The results were plotted as mean ± standard deviation of the mean in the graph (GraphPad Prism 6, Inc., San Diego, CA, USA).

Quantitative analysis of FAS gene expression using real-time PCR

In order to extract RNA from the ovaries, small tissue samples were prepared. RNA extraction from tissue samples was done with Trizol, and using the reverse transcription reaction, mRNA to cDNA was converted using the manufacturer's instructions (Biofact, South Korea). Then, to determine the quality of cDNA, polymerase chain reaction (PCR) was performed using a specific primer for the beta-actin gene (Metabion, Germany) (Table 1) as a housekeeping gene. cDNA was used as a template for real-time PCR to evaluate changes in FAS gene expression in tissues taken from different groups.

Real-time PCR reaction using FAS gene specific primer (Metabion, Germany) (Table 1) and Power SYBR green PCR master mix (Applied Biosystems, UK) using the Real-time PCR system step one plus device (Applied Biosystems, UK) during a program including a temperature of 95°C for 10 minutes, 40 cycles including a temperature of 95°C for 15 minutes and 60°C for 1 minute were performed. Average CTs were calculated using $2^{-\Delta CT}$.

Gene name	Primer sequence (5'-3')
Fas-Forward	TATCACCACTATTGCTGGAGTCA
Fas-Reverse	GCTGTGTCTTGGACATTGTCA
β-Actin- Forward	CGTGCGTGACATTAAAGAGAA
β-Actin- Reverse	CGCTCATTGCCGATAGTGAT

Table 1. Primer sequence of beta-actin and FAS genes used in real-time PCR as Housekeeping and apoptosis markers.

Results

FAS gene expression level findings

Chart 1 shows the mean and standard deviation of FAS gene expression level in different groups. In the AA group, FAS gene expression level increased significantly compared to the control group ($p<0.05$), however, no significant difference was found in the FAS gene expression level in the NAC group with the control group ($p>0.05$). FAS gene expression level in AA+NAC1 and AA+NAC2 groups increased significantly compared to the control group ($p<0.05$). On the contrary, in both groups, a significant decrease in FAS gene expression was observed compared to the AA group ($p<0.05$). In the AA+NAC3 group, there was a significant decrease in FAS gene expression compared to the AA group ($p<0.05$), while there was no significant difference with the control group ($p>0.05$).

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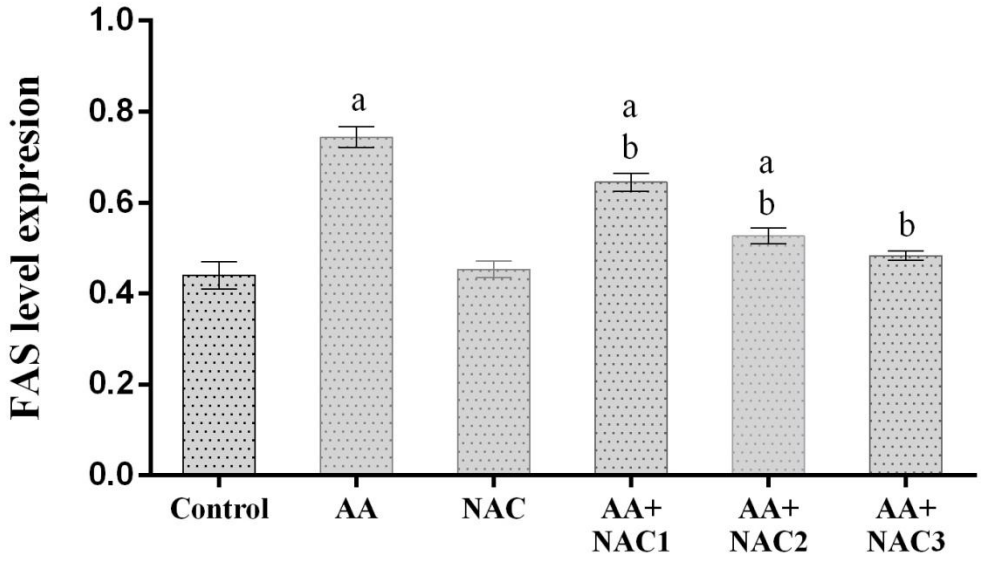

Graph 1. Comparison of mean and standard deviation of FAS gene expression level in different groups. a: compared to the control group. b: Compared with AA group.

Discussion

The results of this study showed that acrylamide with a dose of 50 mg/kg can induce apoptosis in rat ovarian tissue by increasing FAS gene expression. On the other hand, N-acetylcysteine, especially at a dose of 40 mg/kg, was able to inhibit apoptosis induced by acrylamide by reducing the FAS gene expression level in rat ovarian tissue. Oxidative stress is the result of increased ROS, which includes anions, superoxide, hydrogen peroxide, and hydroxyl radicals. ROS can degrade cellular macromolecules of proteins, lipids and DNA and finally induce cell death through apoptosis. There are two main pathways of apoptosis. 1- The extrinsic pathway is stimulated by FAS and the tumor necrosis factor (TNF) family through death receptors and leads to the activation of caspase 8. 2- The intrinsic pathway starts with the release of cytochrome *c* from the mitochondria and leads to the activation of caspase 9, which then cleaves procaspase 3 to activate caspase-3 (Chen *et al.,* 2013). It has been proven that acrylamide induces apoptosis in various tissues of laboratory animals (Yousef & El-Demerdash, 2006; Zhu *et al.,* 2008). It has been reported that long-term exposure to acrylamide affects the expression of apoptosis-related proteins (Li *et al.,* 2006). ROS production by acrylamide leading to cell damage has been reported in several studies (Naruszewicz *et al.,* 2009; Yousef & El-Demerdash, 2006). In this study, Acrylamid increased the expression level of FAS, which seems that Acryl Omid exerts its toxic effects on the ovarian tissue of rats through the extrinsic pathway of apoptosis. The results of our study are consistent with the results of other studies (Camacho *et al.,* 2012; Shahrzad *et al.,* 2021). Previous studies have shown that acrylamide can cause oxidative stress in vitro and in vivo (Yousef & El-Demerdash, 2006; Pan *et al.,* 2015). Oxidative stress is due to an imbalance between the systemic production of ROS and the antioxidant defense against free radicals (Halliwell, 2006; Ott *et al.,* 2007). ROS plays an important physiological role in triggering changes under intracellular and extracellular conditions (Martindale & Holbrook, 2002). ROS levels have been shown to increase in a time- and dose-dependent manner after exposure to acrylamide (Rodriguez-Ramiro *et al.,* 2011). In addition, malondialdehyde, an important indicator of lipid peroxidation damage, has been reported to increase in cells treated with acrylamide. The antioxidant glutathione is vital for scavenging free radicals such as ROS. Acrylamide reduces the antioxidant capacity of cells by reducing glutathione levels, which may be due to the

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combination of acrylamide and its main metabolite, glycidamide, with glutathione. Therefore, excessive production of ROS and malondialdehyde can cause oxidative damage and eventually induce apoptosis in various tissues (Friedman, 2003).

N-acetylcysteine is an aminothiol and a synthetic precursor of glutathione intracellular cysteine, which is considered as an important antioxidant. Due to its antioxidant role, N-acetylcysteine is widely used to investigate the role of ROS in the induction of apoptosis. The function of N-acetylcysteine is due to its antioxidant property or the inhibition of free radicals by increasing the level of intracellular glutathione (Sun, 2010). Although several in vivo studies have shown that N-acetylcysteine significantly prevents or inhibits oxidative stress under certain conditions, the molecular mechanism of this action of N-acetylcysteine is still not fully understood. The antioxidant activity of N-acetylcysteine can be related to at least three different mechanisms, especially in vivo: 1-Direct antioxidant effect. N-acetyl cysteine can have an antioxidant effect against certain oxidant species. 2- Indirect antioxidant effect. N-acetylcysteine can act as a precursor of cysteine and glutathione. Glutathione is an important and well-known antioxidant that is a substrate of several antioxidant enzymes. 3- The effect of fracture on disulfides and the ability to recover thiol sources, which in turn regulates the redox state (oxidation and reduction) (Aldini *et al.,* 2018). It is generally assumed that the action of N-acetylcysteine is due to its antioxidant property or inhibition of free radicals through increasing the level of intracellular glutathione (Sun, 2010; Tenório *et al.,* 2021). Recently, it has been proven that N-acetylcysteine prevents apoptosis by maintaining intracellular glutathione content and reducing mitochondrial membrane depolarization. It has been reported that N-acetylcysteine can reverse the increased expression of FAS, caspase-3 and caspase-8 in pig intestinal epithelial cells and prevent the induction of apoptosis (Zhu *et al.,* 2013). Also, since thiol modulates the activity of many enzymes, it has been shown that N-acetylcysteine is able to modulate the activity of the enzyme responsible for the cleavage of FAS and caspase-3 (Juric *et al.,* 2009; Delneste *et al.,* 1996). Previous studies have shown that treatment of H9c2 cells with N-acetylcysteine has no effect on FAS or FASL expression levels. This suggests that N-acetylcysteine treatment does not activate Fas-mediated apoptosis in H9c2 cells (Liu *et al.,* 2017). In the present study, the administration of N-acetylcysteine in the NAC group did not change the expression level of FAS compared to the control group, which is consistent with previous studies.

The limitations of this study include the lack of measurement of oxidative stress factors such as malondialdehyde, antioxidant factors such as catalase, superoxide dismutase and glutathione peroxidase, and the expression level of other genes involved in apoptosis such as Bcl-2. Therefore, it is suggested to consider the above cases in future studies.

Conclusion

The results of this study showed that acrylamide with a dose of 50 mg/kg can increase the FAS gene expression level and induce apoptosis in rat ovarian tissue. On the other hand, N-acetylcysteine (especially at a dose of 40 mg/kg) was able to decrease the level of FAS gene expression in rats treated with acrylamide and inhibit apoptosis. Therefore, it seems that N-acetylcysteine, as an antioxidant, can counteract the apoptosis induced by acrylamide and modulate the expression level of genes involved in the process of apoptosis, such as FAS.

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Conflict of interest

No conflict of interest has been declared by the authors.

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تاثیر حفاظتی ان-استیل سیستئین بر سطح بیان ژن FAS در تخمدان موشهای صحرایی تیمار شده با آکریل آمید

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چکیده

زمینه و هدف: ان-استیل سیستئین یک داروی آنتی اکسیدان است که برای درمان اختلالات متعدد از جمله درمان مسمومیتها استفاده می‌شود. این مطالعه با هدف بررسی اثر محافظتی دوزهای مختلف ان-استیل سیستئین بر تغییر سطح بیان ژن FAS در بافت تخمدان موشهای صحرایی تیمار شده با اکریل آمید بعنوان یک ترکیب سمی انجام شد.

مواد و روش‌ها: ۳۶ موش صحرایی ماده بالغ از نژاد ویستار به صورت تصادفی در ۶ گروه مساوی تقسیم شدند. دوز آکریل آمید ۵۰ mg/kg و دوزهای ان-استیل سیستئین ۱۰، ۲۰ و ۴۰ mg/kg بود. گروه بندی حیوانات شامل گروه کنترل، آکریل آمید، ان-استیل سیستئین و ترکیبی از آکریل آمید و ان-استیل سیستئین بود. ۲۸ روز بعد، تخمدان حیوانات خارج گردید و سطح بیان FAS mRNA به روش real-time PCR اندازه‌گیری شد.

یافته‌ها: آکریل آمید به تنهایی باعث افزایش سطح بیان FAS در مقایسه با گروه کنترل گردید ($p<۰/۰۵$) در مقابل، ان-استیل سیستئین در دوز حداکثر (۴۰ mg/kg) تفاوت معناداری را در سطح بیان FAS در مقایسه با گروه کنترل نشان نداد ($p>۰/۰۵$).

نتیجه‌گیری: تجویز آکریل آمید در موشهای صحرایی سطح بیان FAS را در بافت تخمدان افزایش می دهد و آپاپتوزیس را القا می‌کند. از طرف دیگر، تجویز ان-استیل سیستئین در دوز حداکثر (۴۰ mg/kg) سطح بیان FAS را با اثرات آنتی اکسیدانی بهبود می دهد و آپاتوزیس القا شده را در بافت تخمدان مهار می کند.

واژه‌های کلیدی: *ان-استیل سیستئین، آکریل آمید، تخمدان، ژن FAS، موش صحرایی*

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