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Mobile phone radiation might alter gene expression in the oral squamous epithelial cells



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Abstract

Background: Accumulating evidence has shown that radiofrequency radiation (RFR) emitted by mobile *phones* is a potential factor for DNA damage. Whether RFR affects *the* gene expression of human genes still requires further research. This may help in understanding the mechanisms of action of this radiation. On the assumption that expression of BAMBI and Survivin in the oral squamous epithelial cells might be modified in response to RF electromagnetic field (RF-EMF) exposure, the current study was conducted on a group of young university student volunteers.

Results: Statistical analysis of the RT-PCR data indicated that no significant association (P value > 0.05) exists between the expression of either *gene*, and neither the length of history nor the frequency of the phone use.

Conclusions: Although no clear RF-EMF signature on gene expression could be detected in this in this preliminary study, it is one of the few studies indicating that molecular-level changes might take place in *humans* in response to chronic mobile phone EMR exposure. Further investigations in this field are warranted.

Keywords: BAMBI gene, Gene expression, Mobile phone, Oral squamous epithelial cells, Radiofrequency radiation, Survivin

Background

The *impact* of radiofrequency electromagnetic radiation (RF-EMR) on living cells is a relatively new avenue of interest with respect to human health. The exact mechanism of action by which EMR emitted from mobile phones interferes with biological functions is still not fully understood. Recently, it has been found that although different brands of smartphones exhibited cytotoxicity in the oral squamous epithelial cells, they do not appear to represent risks of inducing genetic toxicity [1]. Moreover, comet and TUNEL assays have been employed to determine DNA damage and rate of apoptosis, respectively [2]. The numbers of apoptotic *oral* squamous epithelial cells have been highest in medium (30-60 min/day) phone users. However, no strong link has been found between the degree of apoptosis and cumulative years of mobile phone use. Currently, only a few published human studies have investigated alterations in gene/protein expression

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in response to *direct* exposure to radiofrequency radiation (RFR). Contradictory results have been reported by different in vivo and in vitro experiments in various types of cells using a variety of proteomic and transcriptomic techniques. In the hippocampus of Wistar *rats*, it has been indicated that RFR may lead to profound epigenetic modifications, which in turn may cause alterations in gene expression [3]. Proteomics techniques have proved that human endothelial cell line EA.hy926 [4] and lens epithelial cells [5] show alterations in protein expression following exposure to RF-EMF. Contrary to this, exposure of breast cancer cell line (MCF-7) to 1800 MHz RF-EMFs had no significant effect on protein expression [6]. Data collected from in vivo studies proposed that human skin cells may respond to RF-EMF by changing protein expression *profiles* [7].

Survivin is a member of the human eight protein family known to prevent apoptosis [8]. This protein interacts in a unique fashion with components of the mitotic spindle apparatus that regulates cell division [9-11]. Previous research [12, 13] has suggested that Survivin is an ideal



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candidate marker for the diagnosis and prognosis of cancers. Expression of Survivin in tumors has been *linked* not only to inhibition of apoptosis, but also resistance to chemotherapy and invasiveness of malignant cells [10]. Survivin affects key mechanisms of the immune system, including the stimulated proliferation of T cells, differentiation of CD4⁺ T cells and hemostasis of CD8⁺ memory T *cells* [14]. There have been prominent advances in the last few years in targeting Survivin through small molecule inhibitors or immunotherapy.

BAMBI (the bone morphogenetic protein (BMP) and activin membrane-bound inhibitor) is an evolutionally conserved gene in vertebrates [15]. It was first described for its role in the formation and turnover of bone. Later, attention has been directed to the potential involvement of BAMBI in inflammatory response [10, 16–19]. BAMBI encodes a transmembrane glycoprotein related to the type I receptors of the transforming growth factor-beta (TGF-beta) family [17]. It is known that the members of this family play crucial roles in signal transduction in many developmental and pathological processes. Human BAMBI may act as a molecular switch to control TGF- β signaling strength and interleukin-17-producing effector T helper/regulatory T (Th17/Treg) cell balance. Thus, it may be utilized not only as a biomarker but also as a target of new treatment protocols that maintain immune tolerance [20]. The overexpression of BAMBI has been linked with the suppression of the response of cancer cells to TGF- β signaling pathway, to inhibit aggressiveness and autophagy of several types of tumors [21]. Additionally, BAMBI has been identified as a hallmark of non-small cell lung cancer which possesses antitumor potentiality [22].

Both Survivin [14] and BAMBI [20] are cytokine *apop-tosis-related* genes. While Survivin inhibits apoptosis [10, 11], BAMBI suppresses autophagy [21]; thus, both are involved in the regulation of cell division. In addition, both are promising biomarkers in the new treatment protocols of immunotherapy. In the present study, it was hypothesized that exposure to RFR emitted from the *mobile phones* during calls is associated with the expressions of the two genes in the oral squamous epithelial cells. This type of cells is shed from tissues usually exposed to radiation during mobile calls. Knowledge of the mechanism of action of RF-EMFs will be of great practical value in view of the tremendous increase in the use of mobile phones and the installation of mobile phone towers.

Methods

Study population

This *cross-sectional* study has been carried out on randomly recruited 48 volunteer university students (24 males, 24 females) aged between 18 and 27 years. This age range was selected because it is expected that this young population group is exposed to daily mobile phone use. In short-term exposure, people are getting exposed to RF-EMR for 10, 15, 30 min or 1 h. In longterm exposure, the duration increases up to 5-6 h or even longer than this [23]. It has been reported that, on average, the person spends 90 min a day on his/ her phone [2]. Written informed consent has been signed by every participant. Prior to providing the oral mucosal cells, each person was interviewed by the same researcher to standardize data *collection*. The specially constructed detailed questionnaire included information on age, gender, type of mobile used, frequency of mobile phone use (min/day) and the history of mobile use (number of years). The study has excluded smokers, or people who received drug therapy during the last 3 months, suffering any disease including cancer, exposed to radiotherapy including dental procedures, dietary supplements and regular mouthwash users. All subjects completed the course of the study. The research staff was ready to answer any query that may arise at any time during the study. Participants were divided into three groups based on the frequency and intensity of phone use: low mobile phone users (less than 30 min/day), medium mobile phone users (30-60 min/day) and heavy mobile phone users (more than 60 min/day). The participants were further categorized into three classes on the basis of the history of phone use: short-, medium-, and long-duration users for those who used their phones for periods shorter than 5 years, 5-10 years and longer than 10 years, respectively. Appropriate Ethical Committee approval and the informed consent of all participating subjects were obtained. It was not possible to find age-matched non-users as a control group because the wide usage of mobile phones is so profound; today each and every person uses a cell phone.

Cell samples

Participants were requested not to eat or drink 1 h prior to collecting the oral squamous epithelial cells. The oral cavity was rinsed with drinking water. Two samples were obtained, at the same time of the day; between 10 a.m. and 12 noon, from the inner surface of both sides of the *donor's* cheeks using sterile, *small-headed* plastic toothbrushes. To dislodge cells and release them, the brush was immersed in a buffered medium with repeated rotation. The collected material was centrifuged (*Sigma Cold Centrifuge, Osterode am Harz, Germany*) for 10 min at 14,000 rpm, and the cell pellets were harvested for RNA isolation.

Total RNA extraction

The total RNA of the oral squamous epithelial cells was extracted by TRIzol reagent (Invitrogen, California, USA) according to the manufacturer's instructions. The cells were placed in 1.5-ml Eppendorf tubes and centrifuged at 14,000 rpm for 10 min. The pellets were washed in Dulbecco's modified Eagle medium (Euroclone group, EU) and re-centrifuged at 14,000 rpm for 10 min. The supernatant was aspirated, and 200 µl TRIzol was transferred to each tube. The cell suspensions were then vortexed for 1 min and left for incubation at room temperature (RT) for 5 min. After that, 0.2 ml chloroform/1 ml volume TRIzol was added and the tube was votexed for 30 s. The supernatant was transferred to a new tube, and 0.5 ml isopropanol and 50 µl 0.3 M sodium acetate were added to a volume of 1 ml supernatant. The contents were stored at -20 °C for 20 min. After centrifugation at 15,000 rpm for 5 min, the resulting RNA pellets were washed with 70% ethanol. Cells were centrifuged again at 15,000 rpm at 4 °C for 3 min and washed with absolute ethanol 2 times, mildly dried and dissolved in 30 µl N.F water. The latter step was repeated, and the cells were dried for 10 min. The pellets were suspended in 30 µl N.F water. Finally, the sample's RNA concetration was measured using Thermo Fisher Scientific NanoDrop (Vantaa, Finland), and the RNA sample was stored at -80 °C or processed directly for cDNA synthesis.

DNase treatment protocol

The prepared RNA samples were treated with DNase using New England BioLabs DNase kit (Ipswich, Massachusetts, USA). Briefly, 10 μ l of the RNA sample were mixed with 10 μ l of DNase I reaction buffer 1X and 1 μ l of RNase-free DNase I. The volume was completed up to 100 μ l with N.F water. After mixing, the components of the mixture were incubated at 37 °C for 10 min, and then 1 μ l of 0.5 M EDTA was added to the mixture and heated to 75 °C for 10 min. After cooling of the tube contents, DNA-free RNA was extracted using the protocol mentioned in the above section.

RT-PCR analysis

RNA was converted into single-stranded cDNA according to the High-Capacity cDNA Reverse Transcription Kit by *Thermo* Fisher Scientific (Waltham, Massachusetts, USA). A 10 μ l mixture was transferred to a PCR tube along with a 10 μ l of the RNA sample and incubated in Esco Swift MaxPro thermal cycler (Changi, Singapore) under the following conditions: 25 °C for 10 min, 37 °C for 10 min and 85 °C for 5 min. The cDNA sample was diluted 1:1 with F.N water and stored at - 80 °C. The PCR reaction mixture consisted of 1 μ l sample cDNA and 10 µl of QuantiFast 2X Master Mix (Qiagen, Germany) including SYBR GREEN, 2 µl of the related primers and 5 µl N. F water. After mixing, the 20 µl mixtue was transferred to a PCR tube and DNA was amplified. The procedure was carried out under the following conditions: initial denaturation phase, 95 °C for 30 s, followed by 40 cycles with denaturation at 95 °C for 10 s, annealing at 60 °C for 30 s, and the extension phase was performed at 72 °C for 40 s and 72 °C for 2 min. To avoid any inter-run variation, cDNA prepared from human periodontal ligament (PDL) fibroblasts was always run together within one run. Changes in the expression of each target gene were normalized relative to the mean critical threshold (CT) values of glyceraldehyde-3-phosphate dehydrogenase (GAPDH) as a housekeeping gene. The used primers in PCR description were as follows: Survivin: F: 5'-AGAACTGGCCCTTCTTGGAGG-3', R: 5'-CTTTTT ATGTTCCTCTATGGGGTC-3' [13]; BAMBI: F: 5'-CGA TGTTCTCTCTCCCCAG-3', R: 5'-AATCAGCCC TCCAGCAATGG-3' [24]. The sequences of GAPDH primers were: F: 5'-CGCATGGACTGTGGTCATGA-3', R: 5'-TTCACCACCATGGAGAAGGC-3' [25]. The relative expressions of cytokine genes were calculated using comparative Ct $(2^{-\Delta\Delta C}_{T})$ analysis methods and assayed by the CFX96 QPCR machine (BioRad, USA), as in the following equations:

Relative expression =
$$2_T^{-\Delta\Delta C}$$

 $\Delta\Delta Ct = \Delta Ct$ (buccal sample)
 $-\Delta Ct$ (PDL sample)
 $\Delta Ct = AVG.Ct$ (gene of interest)
 $-AVG.Ct$ (housekeeping gene, GAPDH)

Data analysis

Data collection and results analysis were completed under blind code. The results are expressed as mean \pm SEM. Statistical analysis was carried out with the SPSS 19 package (SPSS Inc., Chicago, USA). Differences between exposed groups were analyzed using independent Student's *t* test, while multiple comparisons among more than two groups (as per addiction habits and call duration) were made by Tukey's multiple comparison test.

Results

Analysis of the information collected from the study questionnaire has shown that the 48 respondents used mobile phones whose emitted radiation intensity ranged from 0.92 to 1.16 W/kg. Because there were no statistically significant differences in the records of the male and female users, the results were pooled. The RT-PCR amplification curves for the genes covered are shown in



Fig. 1 Comparative quantification of mRNA expression expressed as real-time PCR amplification curves obtained by plotting fluorescence data against their cycle number



GAPDH in the *center-left* peaks for BAMBI and right peaks for Survivin. The melting curves are displayed as the first negative derivative of the fluorescence versus the temperature. Thus, a peak can be seen at the melting temperature

Table 1 Relative expression of BAMBI and Survivin in oralsquamous epithelial cells from mobile phone users

Group	Number of participants	BAMBI±SEM	P value	Survivin \pm SEM	P value
Total daily call time (min)					
< 30	27	97.9 ± 19.4	0.31	63.9 ± 25.0	0.23
30–60	12	134.9 ± 41.4		13.9 ± 5.3	
>120	9	60.4 ± 29.7		14.4 ± 9.5	
Duration of phone use (years)					
< 5	23	97.1 ± 18.9	0.08	27.5 ± 17.8	0.37
5-10	15	63.3 ± 20.4		38.9 ± 19.7	
>10	10	162.2 ± 53.1		80.7 ± 48.0	

Fig. 1. Melting curves have been generated to confirm the specificity of the RT-PCR reactions (Fig. 2).

A clear view of the melting dynamics of different PCR products has been observed. Low or no detectable differences have been noted in the expression of mRNA of Survivin and BAMBI from the oral squamous epithelial cells from the experimental groups (Table 1, Figs. 3, 4).

Discussion

The main goal of the present study was to assess the possible effect of mobile phones on gene expression levels of Survivin and BAMBIN in 48 mobile phones users. Our data suggest that there has been no significant association (P value[>]0.05) between RFR exposure conditions (frequency and exposure duration) and the expression of Survivin.

The absence of significant gene expression could be explained by the fact that the effects have very low amplitude and therefore are not obvious. It should be remembered that the findings of different studies should be compared with caution. The results of the present experiments on gene expression are in agreement with the finding that Survivin is weakly expressed in most normal differentiated cells [26]. On the other hand, due to its well-characterized function in the inhibition of apoptosis and control of the mitotic apparatus, different researchers have confirmed that Survivin is very strongly expressed during embryonic stages as well as in almost all types of human cancers [10, 26-28]. In addition, the selection of the human cell type to evaluate exposure to mobile phone-emitted radiation is of utmost importance since changes in protein expression in one cell type do not necessitate that similar response will take place. In the available literature, some but not all human cells responded to increases in the expression of genes that encode ribosomal proteins [29]. The latter in vitro study has proved that the same cell type may react differently depending on exposure conditions. The data that have been reported by another research team [30] have confirmed that induction of imbalance in the oxidative status in favor of prooxidants may lead to a significant activation of Survivin expression. This may suggest that oxidative stress could be a critical player in the initiation and development of carcinogenesis. Optimizing knock-out strategies for Survivin in suitable preclinical models have been used to follow up the impact of Survivin blockage on radiation sensitivity [31].

The present results may indicate that exposure of phone users to RFR could be linked to functionally expressed BAMBI levels. It has been pointed out that the modulation of BAMBI expression is cell type-dependent [20]. Unfortunately, however, data on cell type-specific expression of BAMBI are not available. High levels of BAMBI expression have been observed in stimulated CD4⁺ T cells from human peripheral blood [20]. BAMBI expression was limited to endothelial cells of the glomerular and some peritubular capillaries and of arteries



and veins in both murine and human kidneys [16, 32]. Its expression is under the control of lysosomal/autolysosomal degradation.

One point of strength of the present study is the fact that it has been conducted on both sexes. However, several restrictions are worth mentioning: first, the differences in the lifestyle among participants added to other confounding factors that were out of control. These factors may have contributed to different amounts of energy absorption by the cells and consequently the response of these cells. Our analysis did not include all confounding variables due to the relatively small number of participants. Some biases may be inherent in this type of analysis, and we acknowledge this is a limitation.

Conclusions

This study has been designed to survey the potential impact of exposure to RFR emitted from phones during use in the regulation of the activity Survivin and BAMBI in buccal cells. It represents the start of a new approach to studying the health effects of RFR on vital genes. Exposure may induce some expression of BAMBI but may not exhibit a detectable correlation with Survivin expression. Thus, no clear RF-EMF signature on gene expression could be confirmed. The data of the present investigation should be interpreted with care. To arrive at more confirmatory conclusions, sample size should be larger and with people of various age-groups covering different geographical regions. A sample of deaf people may serve as a negative control



for mobile phone users. Since the expression of BAMBI [33] and Survivin [11] is under the control of micro-RNAs (miRNAs), further studies investigating the expression of these molecules among phone users may prove useful. The overall message is that mobile phones should not be used for longer duration so as to avoid damage to DNA.

Abbreviations

BAMBI: BMP and activin membrane-bound inhibitor; BMP: Bone morphogenetic protein; GAPDH: Glyceraldehyde-3-phosphate dehydrogenase; RF-EMR: Radiofrequency electromagnetic radiation, radiofrequency radiation; TGF: Transforming growth factor.

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Not applicable.

Authors' contributions

AMK contributed to concepts, experimental design, data interpretation, literature search, manuscript preparation and manuscript review; KMA helped

in concepts, experimental design, experimental studies, statistical analysis and manuscript review; IFA was involved in experimental studies, data acquisition, statistical analysis, literature search and manuscript review; MAO contributed to experimental studies, data acquisition, manuscript preparation and manuscript review. All authors have read and approved the manuscript before its submission.

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Availability of data and materials

The data generated and/or analyzed during the present study are not publicly available due to participants' privacy but are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate

This cross-sectional study was approved by Human Research Ethics Committee (IRB number 451/2442). The informed consent to participation and for publication was obtained from all subjects.

Consent for publication

Not applicable.

Competing interests

The authors declare that this research was conducted in the absence of any commercial or financial relationships that could be construed as a potential competing interest.

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References

- Prado PGS, Santos IS, Santos MALD et al (2020) Evaluation of genotoxic and cytotoxic effects between different smartphone brands in the oral mucosa epithelium. Oral Surg Oral Med Oral Pathol Oral Radiol 130:e273–e4
- Khalil AM, Alemam IF, Al-Qaoud KM (2020) Association between mobile phone using and DNA damage of epithelial cells of the oral mucosa. J Biomed Biotechnol 3:050–066
- Kumar R, Deshmukh PS, Sharma S, Banerjee BD (2021) Effect of mobile phone signal radiation on epigenetic modulation in the hippocampus of Wistar rat. Environ Res 192:10297
- Nylund R, Leszczynski D (2006) Mobile phone radiation causes changes in gene and protein expression in human endothelial cell lines and the response seems to be genome- and proteome-dependent. Proteomics 6:4769–4780
- Li HW, Yao K, Jin HY et al (2007) Proteomic analysis of human lens epithelial cells exposed to microwaves. Jpn J Ophthalmol 51:412–416
- Zeng Q, Chen G, Weng Y et al (2006) Effects of Global System for Mobile Communications 1800 MHz radiofrequency electromagnetic fields on gene and protein expression in MCF-7 cells. Proteomics 6:4732–4738
- Karinen A, Heinävaara S, Nylund R, Leszczynski D (2008) Mobile phone radiation might alter protein expression in human skin. BMC Genom 9:77
- Li F (2003) Survivin study: what is the next wave? J Cell Physiol 197:8–29
 Fortugno P, Wall NR, Giodini A et al (2002) Survivin exists in immuno-
- chemically distinct subcellular pools and is involved in spindle microtubule function. J Cell Sci 115:575–585
- 10. Garg H, Suri P, Gupta JC, Talwar GP, Dubey S (2016) Survivin: a unique target for tumor therapy. Cancer Cell Int 16:49
- 11. Rahban D, Mohammadi F, Alidadi M et al (2019) Genetic polymorphisms and epigenetic regulation of survivin encoding gene, BIRC5, in multiple sclerosis patients. BMC Immunol 20:30
- 12. Span PN, Sweep FC, Wiegerinck ET et al (2004) Survivin is an independent prognostic marker for risk stratification of breast cancer patients. Clin Chem 50:1986–1993
- Shen C, Hu L, Xia L, Li Y (2008) Quantitative real-time RT–PCR detection for Survivin, CK20 and CEA in peripheral blood of colorectal cancer patients. Jpn J Clin Oncol 38:770–776
- 14. Ebrahimiyan H, Aslani S, Rezaei N, Jamshidi A, Mahmoudi M (2018) Survivin and autoimmunity; the ins and outs. Immunol Lett 193:14–24
- 15. Onichtchouk D, Chen YG, Dosch R et al (1999) Silencing of TGF-beta signaling by the pseudoreceptor BAMBI. Nature 401:480–485
- Xavier S, Gilbert V, Rastaldi MP et al (2010) BAMBI is expressed in endothelial cells and is regulated by lysosomal/autolysosomal degradation. PLoS ONE 5:e12995
- 17. Zhang JC, Chen G, Chen L et al (2016) TGF-beta/BAMBI pathway dysfunction contributes to peripheral Th17/Treg imbalance in chronic obstructive pulmonary disease. Sci Rep 6:31911
- Yang Y, Guo C, Liao B et al (2017) BAMBI inhibits inflammation through the activation of autophagy in experimental spinal cord injury. Int J Mol Med 39:423–429
- 19. Sun S-W, Chen L, Zhou M et al (2019) BAMBI regulates macrophages inducing the differentiation of Treg through the TGF- β pathway in chronic obstructive pulmonary disease. Respir Res 20:6
- Liu HJ, Chen G, Chen L et al (2018) Cytokine-induced alterations of BAMBI mediate the reciprocal regulation of human Th17/Treg cells in response to cigarette smoke extract. Int J Mol Med 42:3404–3414
- Yuan CL, Liang R, Liu ZH et al (2018) Bone morphogenetic protein and activin membrane-bound inhibitor overexpression inhibits gastric tumor

cell invasion via the transforming growth factor- β /epithelial-mesenchymal transition signaling pathway. Exp Ther Med 15:5422–5430

- 22. Wang X, Li M, Hu M, Wei P, Zhu W (2017) BAMBI overexpression together with β -sitosterol ameliorates NSCLC via inhibiting autophagy and inactivating TGF- β /Smad2/3 pathway. Oncol Rep 37:3046–3054
- Renke A, Chavan M (2018) Study of adverse health effects due to mobile tower radiation situated in densely populated residential areas. Int J Future Revol Comput Sci Commun Eng (IJFRCSCE) 4:63–67
- Pfeifer CG, Karl A, Berner A et al (2016) Expression of BMP and actin membrane-bound inhibitor is increased during terminal differentiation of MSCs. Stem Cells Int. Article ID 2685147
- Hoyng S, Gnavi S, de Winter F et al (2014) Developing a potentially immunologically inert tetracycline-regulatable viral vector for gene therapy in the peripheral nerve. Gene Ther 21:549–557
- 26. Adida C, Crotty PL, McGrath J et al (1998) Developmentally regulated expression of the novel cancer anti-apoptosis gene Survivin in human and mouse differentiation. Am J Pathol 152:43–49
- 27. Vachtenheim J, Vlckova K (2016) Insights into the regulation of Survivin expression in tumors. Single Cell Biol 5:139
- 28. Wheatley SP, Altieri DC (2019) Survivin at a glance. J Cell Sci 132:jcs223826
- 29. Remondini D, Nylund R, Reivinen J et al (2006) Gene expression changes in human cells after exposure to mobile phone microwaves. Proteomics 6:4745–4754
- Maxia C, Perra MT, Demurtas P et al (2009) Expression of Survivin protein in pterygium and relationship with oxidative DNA damage. J Cell Mol Med 13:207–209
- 31. Hanif A, Lee S, Gupta M et al (2020) Exploring the role of Survivin in neuroendocrine neoplasms. Oncotarget 11:2246–2258
- Lai H, Chen A, Cai H et al (2020) Podocyte and endothelial-specific elimination of BAMBI identifies differential transforming growth factor-β pathways contributing to diabetic glomerulopathy. Kidney Int 98:601–614
- Huang K, Shi X, Wang J et al (2019) Upregulated microRNA-106a promotes porcine preadipocyte proliferation and differentiation by targeting different genes. Genes 10:805

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