Journal of Health Study and Medicine

2021, No. 2

Redaktor naczelny / Editor in chief
dr n. med. Joanna Sułkowska

Skład / Adjustment, Typesetting
Witold Kowalczyk

ISSN 2451-1471

ul. Kilińskiego 109
90-011 Łódź
tel./fax: (042) 676 25 29 wew. 339
e-mail: wydawnictwo@spoleczna.pl

Wersja elektroniczna publikacji jest wersją podstawową, dostępną na stronie:
jhsm.san.edu.pl
E-version is the original version of the article, available:
jhsm.san.edu.pl

Artykuły recenzowane / All the articles published are subject to reviews.

Journal of Health Study and Medicine
2021, No. 2
pp. 5-23
DOI 10.36145/JHSM2021.07

The Pilot Study of C-jun and TGF-beta Immunoexpression in Relation to the Oldest Cancer Biomarkers – Tumor Histology and Proliferation Rate in Glioblastoma and Neuroblastoma Models of Cancer Disease

Aleksandra Hincz[1]
https://orcid.org/0000-0003-2423-3389

Katarzyna Joanna Gąsior[1]
https://orcid.org/0000-0001-6626-6141

Marta Jadwiga Cichoń[1]
https://orcid.org/0000-0002-7864-9112

Paweł Kapszewicz[2]
https://orcid.org/0000-0002-8438-195X

Elżbieta Sałacińska-Łoś[3]
https://orcid.org/0000-0002-3912-7143

Magdalena Orzechowska[4]
https://orcid.org/0000-0002-8301-8059

Katarzyna Taran[1]
https://orcid.org/0000-0002-0598-9079

[1] Department of Pathomorphology, Medical University of Lodz, Poland
[2] Department of Ophthalmology, Mother's Health Memorial Hospital, Lodz, Poland
[3] Department of Oncology and Pediatric Surgery, Medical University of Lodz, Poland
[4] Department of Molecular Carcinogenesis Lodz, Medical University of Lodz, Poland

Address for correspondence

Katarzyna Taran
Laboratory of Isotopic Fractionation in Pathological Processes
Department of Pathomorphology, Chair of Oncology
Medical University of Lodz
251 Pomorska St., 92-213 Lodz, Poland
katarzyna.taran@umed.lodz.pl

Abstract

Background: *Prognostic factors in cancers are believed to be one of the most important discoveries in oncology. However, due to the development of integrated science they are not restricted to morphological anomalies anymore. In everyday routine the light microscope markers are systematically replaced by new ones, especially of immunohistochemical and molecular type. It leads to the question what is the relation of the oldest prognostic parameters to the basic and newly discovered pathways common for cancer disease itself.*

Objectives: *The aim of presented studies is to investigate the most modern factors involved in pathological processes in the two models of cancer disease, glioblastoma in adults and neuroblastoma in developmental age to search for potential relation with the established prognostic factors appropriately to the tumor type, including the oldest known light microscope parameters – tumor histology and proliferation rate of cancer cells.*

Material and methods: *Immunohistochemical assessment of expression of c-jun in glioblastoma and TGF-beta in neuroblastoma group in relation to chosen histoclinical features: patients' related (age, gender) and tumor related; including all the widely excepted prognostic parameters regarding the tumor type (location, histological type (neuroblastoma, ganglioneuroblastoma, ganglioneuroma), tumor histology (favorable, unfavorable), Ki-67 index, stage.*

Results: *Variations of c-jun immunoexpression were revealed in glioblastoma as well as differences in TGF-beta expression in neuroblastoma regarding the examined histoclinical features. Furthermore, in both cancer groups the levels of the examined protein expression appeared to relate to cancer cell proliferation estimated by the established parameter – Ki67 indices.*

Conclusions: *In both models of cancer disease, glioblastoma in adults and neuroblastoma in developmental age there is a crossing of pathways of the oldest and the newest cancer disease markers. Although integrated science offers the most advanced approaches it is important to consider the old*

established prognostic parameters in prognostication in individual, especially doubtful cases.

Key words: *biomarkers, intercellular signalling peptides and proteins, glioblastoma, neuroblastoma*

Introduction

Glioblastoma (GBM) is grade IV glioma according to WHO and one of the most aggressive neoplasms in humans, characterized by extremely poor prognosis [1]. Although for now several molecular markers are confirmed to be relevant in the development of glioblastoma (isocitrate dehydrogenase (IDH) mutations, MGMT promoter methylation, EGFR amplification/mutations, and vascular endothelial growth factor (VEGF) overexpression, the most characteristic and mandatory feature for the GBM diagnosis is light microscope finding – the proliferation of endothelial cells [2–5]. Angiogenesis in GBM makes this tumor a suitable model of cancer growth as well as highly investigated process for the knowledge of the neoplastic disease itself.. Angiogenesis in GBM makes this tumor a suitable model of cancer growth as well as highly investigated process for the knowledge of the neoplastic disease itself.

Neuroblastoma (NBL) may start in early forms of nerve cells found in a developing embryo and is typical cancer of infancy and early childhood. However, these tumors themselves present complexity of cancer disease from spontaneous regression to unjustified treatment failures and as the group make well known cancer disease model for developmental age [6]. They are also characterized by extremely numerous prognostic parameters: patient's age, tumor histology (favorable histology or unfavorable histology), DNA ploidy, MYCN gene amplifications, chromosome changes: 1p deletions, 11q deletions, 17q gain, Neurotrophin (nerve growth factor) receptors: especially TrkA, serum (blood) levels of ferritin and neuron-specific enolase (NSE) and lactate dehydrogenase (LDH). However, despite their high number of markers the course of the disease is unpredictable in all of the cases.

C-jun and TGF-beta proteins are modern and highly investigated proteins involved in pathological processes, especially cancer disease [7, 8]. It is underlined in both – they are fundamental part in basic pathways characterized cancer itself [9–11].

The role of the c-jun protein that belongs to AP-1 family is to signal--transduce transcription which make it a crucial factor in the cell life cycle [12–14].

TGFβ is believed to be an extremely important factor in the process of tumorigenesis and invasiveness by being involved in evoking oxidative stress that is associated with cancer development [15].

The aim of presented studies was to investigate the expression of above listed factors in two models of cancer disease – glioblastoma in adults and neuroblastoma in developmental age to search for their potential relation with the established prognostic factors appropriately to the tumor type, including the oldest known light microscope parameters - tumor histology and proliferation rate of cancer cells.

Material and methods

The studies were performed according to the agreement of Bioethics Committee of the Medical University od Lodz RNN/350/19/KE and RNN/244/19. Each cancer study group consisted of 20 cases from the archives of Department of Pathomorphology, Chair of Oncology, Medical University of Lodz, Poland. Glioblastoma and neuroblastoma tissue samples were paraffin embedded and these paraffin blocks were sectioned to 4–5 μm samples, routinely stained with hematoxylin and eosin (H+E) and used for further immunohistochemical tests in the PowerVision detection system by ImmunoLogic according to the immunoperoxidase method, with the use of primary rabbit polyclonal antibodies directed against c-jun by Cell Signalling Technology and TGFβ by Biorbyt. Paraffin sections placed on adhesive slides and dried in an incubator at 56°C for 1 hour, were passed to dewaxing in a series of xylenes and alcohols of decreasing concentrations (96%, 80%, 70%, 60%). By the use of 3% solution of perhydrol in methanol for 5 minutes endogenous peroxidase activity could be inhibited. For the purposes of immunohistochemistry, it was required to preserve tissue antigenicity and made it proper for the examined antibodies to work. In order to do that a specific sequence had to

be followed. The sections were heated in Target Retrieval Solution at pH 9.0 DAKO, in a water bath at 95°C for 45 min. After cooling, they were washed twice in 0.05 M TRIS buffer (TBS) at pH 7.6 for 5 min. Then they were subjected to overnight incubation in a refrigerator at 4°C, in a humid chamber, with appropriately diluted antibodies: anti-c-jun 1:50 and anti-TGFβ 1:400.

The next step was to wash it twice in TBS and visualize in the ImmunoLogic PowerVision two-step visualization system to observe the antigen-antibody response. In order to do that the sections were incubated 30-minute in the above-mentioned conditions with a polymer labelled with peroxidase and associated with secondary goat antibodies directed against the used polyclonal rabbit antibodies. The last stage of detection was an enzymatic reaction in which a colored product appeared when the substrate for peroxidase – 3,3` –diaminobenzidine tetrachloride (DAB) was used (incubation time with DAB solution – 2 min.). In the end, when a positive immunohistochemical reaction was achieved, the nuclei were stained with Meyer's hematoxylin (2 min.), and then the sections were dehydrated in a series of alcohols of increasing concentrations (70%, 80%, 96%) and passed through a series of acetones and xylenes. The prepared Histokit was embedded in an anhydrous medium. The negative control of the method were sections in which the primary antibodies were replaced with TBS using the same immunohistochemical procedure. Sections showing a previously known strong positive reaction to the test antigen were used as a positive control. The slides were assessed using a light microscope. Positive reaction was defined as the brown coloration of cell nuclei and presented as an index in each case. Histoclinical analysis of c-jun or tgf-beta expression was done regarding patients' related features (age, gender), tumor related parameters including all the widely excepted prognostic parameters regarding the tumor type (location, histological type – neuroblastoma, ganglioneuroblastoma, ganglioneuroma, tumor histology – favorable or unfavorable), Ki-67 index, stage, lymph node status, distant metastases, recurrences). The obtained data were evaluated in statistical analysis

that was performed with the use of the statistical packages rstatix and ggpubr in the R environment (R 4.0.2). In the first stage, the Shapiro--Wilk test followed by Levene's test were carried out and in the subsequent stages of the analysis, non-parametric Kruskal-Wallis test was used. The hypotheses were tested by applying a non-parametric signed rank Wilcox test or in the case of normally distributed data the t-test. Correlations between parameters were calculated using the Spearman coefficient. The significance level (p) was considered statistically significant when its value was < 0.05.

Results

Glioblastoma study

C-jun immunoexpression was found in all the examined GBM cases and appeared variously with differences in gender, age, and location of the tumor as well as Ki-67 indices (Figure 1).

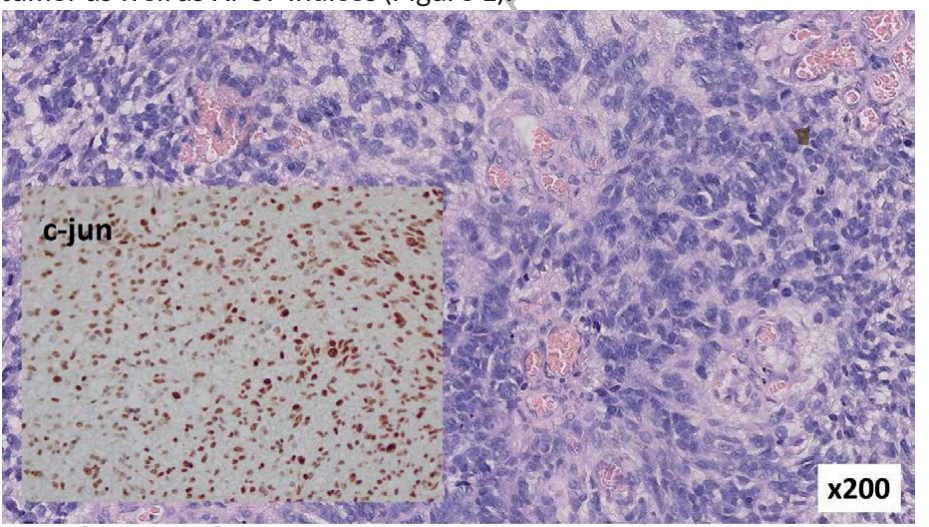

Figure 1. The example of c-jun immunoexpression in GBM (left), the background – proliferation of endothelial cells in GBM, hematoxylin and eosin (H&E) stain, orig. magn. 200x

The c-jun indices appeared as follow:

Among males, the median c-jun expression was 38% (mean = 36.7 ± 17.3), whereas among females it was lower and reached 34% (mean = 32.6 ± 7.57) (Figure 2).

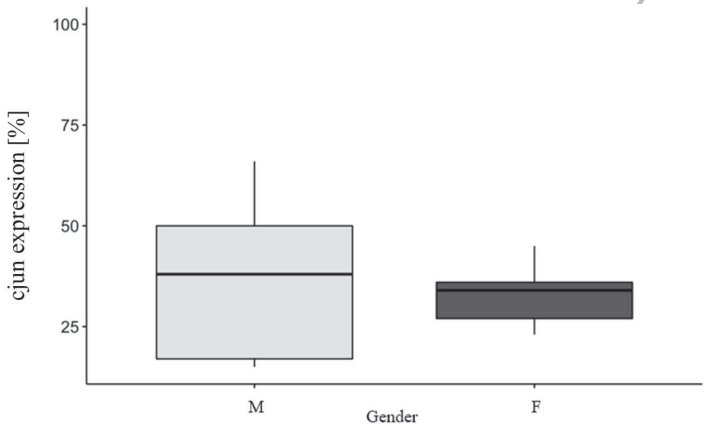

Figure 2. The box-plot of the values of difference of c-jun expression between males (M) and females (F) in GBM group

Median c-jun expression for right hemisphere, left hemisphere, and brain in general was 40.5% (mean = 38.3 ± 16.2), 39.5% (mean=36 ± 15.5), and 32% (mean = 33.1 ± 14.4), accordingly (Figure 3). Differences in c-jun immunoexpression indices listed above do not appear statistically significant.

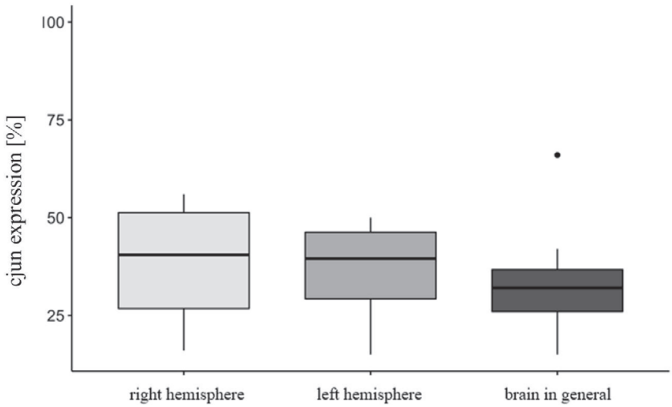

Figure 3. The box-plot of the values of difference of c-jun expression regarding tumor location in GBM group

There was observed a moderate association of c-jun expression and age of women (rho=-0.64) (Figure 4), a strong association of c-jun expression and age and location in left hemisphere (rho = -0.80) (Figure 5), as well as c-jun expression and Ki-67 expression in right hemisphere located tumors (rho = -0.71) (Figure 6).

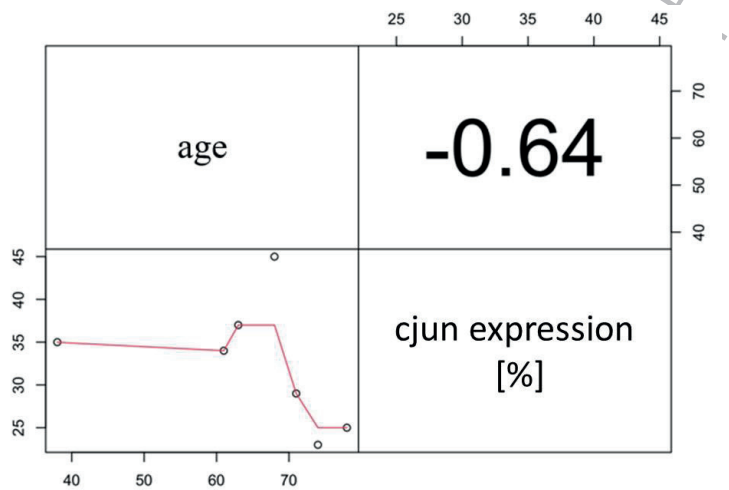

Figure 4. Correlation between c-jun expression and patients age among females – rho Spearman

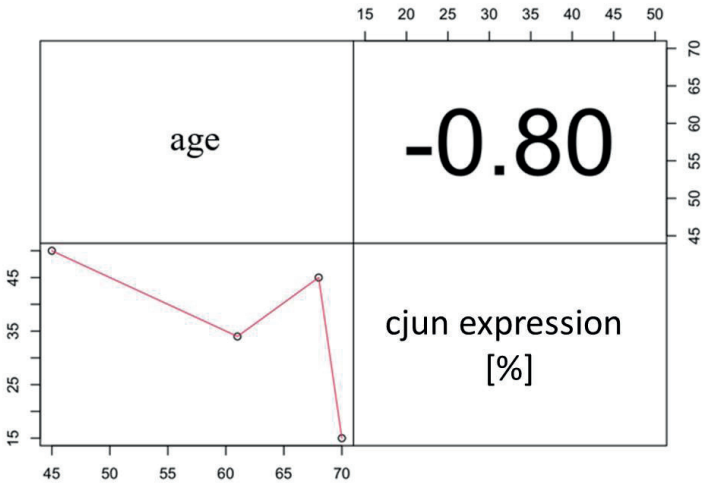

Figure 5. Correlation between c-jun expression, patients age and tumor location in left hemisphere – rho Spearman

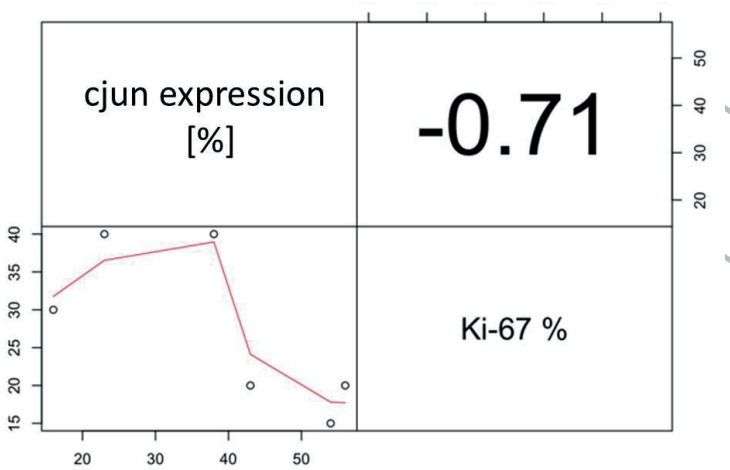

Figure 6. Correlation between c-jun expression, Ki-67 expression and tumor location in right hemisphere – rho Spearman

Neuroblastoma group

TGF-beta immunoexpression was found in all the examined GBM cases and appeared various with differences in gender, age, histological type (neuroblastoma, gangioneuroblastoma or ganglioneuroma), tumor histology (favorable or unfavorable) (Figure 7) .

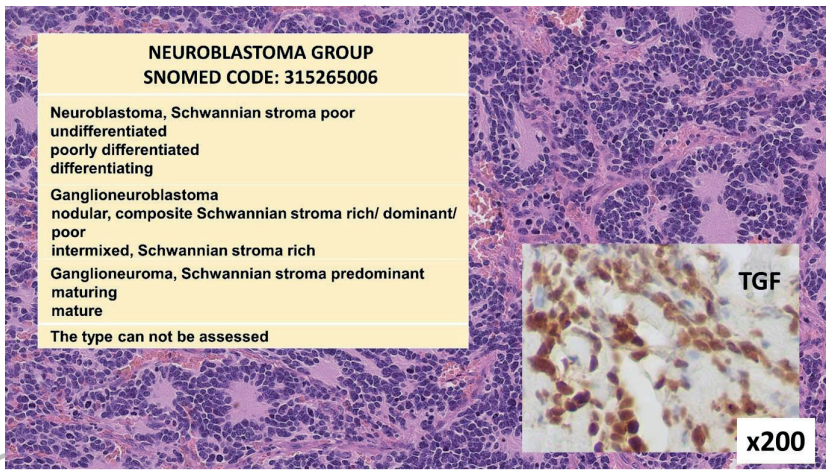

Figure 7. The example of TGF-beta immunoexpression in NBL (right), the background –Schwannian stroma poor, poorly differentiated NBL, hematoxylin and eosin (H&E) stain, orig. magn. 200x

There was revealed statistically significant relation of TGFβ expression in histological types of tumors of neuroblastoma group, p = 0.0024, including tumor histology (favorable and unfavorable), p = 0.0076 (Figure 8).

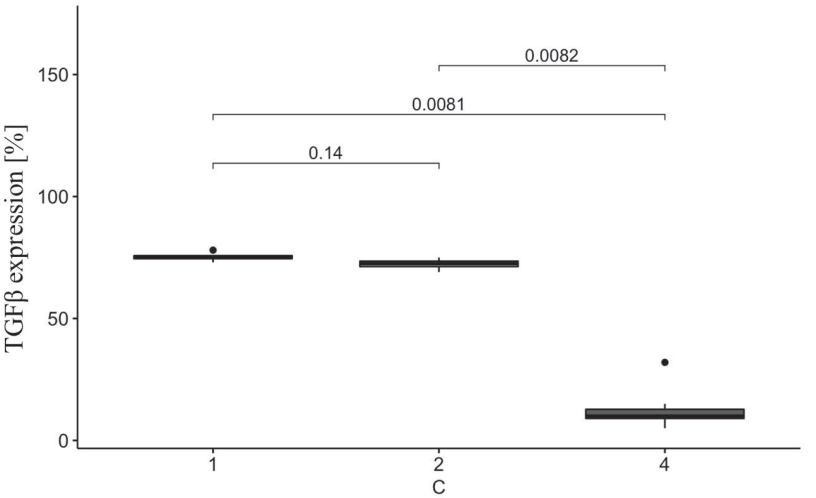

Figure 8. The values of TGFβ expression in NBL group (C) – C1 neuroblastoma, C2 ganglioneuroblastoma, C3 ganglioneuroma

Discussion

The search for universal cancer biomarkers is one of the leading aims of current oncology. Complete understanding of basic pathways of cancer disease seems to be the key to cancer treatment and creating the personalized strategies that will allow to achieve optimal results. Various patterns of c-jun expression in glioblastoma and TGF-beta in neuroblastoma group obtained in presented studies are understandable regarding the fact that both the examined cancers cover the fundamental cancer mechanisms, becoming the models for cancer disease in adulthood and developmental age, appropriately.

C-jun expression is associated with the majority of the occurring processes including proliferation, differentiation and apoptosis [12]. Its up-regulation is commonly seen in the central nervous system and evidences show its connection with many pathologies such as inflammations,

ischemia and neurodegenerative diseases as well as with many physiological reparative processes [13, 14, 16]. It is also noticeable that many studies confirm its involvement in development and progression of human malignancies including glioblastoma [7, 8, 17–19].

Although, the obtained results did not achieve the minimum threshold of statistical significance probably due to the limited study subgroups, where only huge differences within homogeneous groups may be proven, they well illustrate the complexity of GBM biology with non-complex morphology and lack of prognostic significance of morphological findings.

There are very intriguing observed differences in c-jun expression in GBM of the right and left hemisphere, that seem to be worth of further evaluation. Among these findings it is important to underline the correlation of c-jun and Ki-67 expression found in the right hemisphere. The estimation of cancer cell proliferation is one of the oldest prognostic parameters with widely accepted value in many cancer types. Preliminary attempts of investigation of proliferation rate were based on light microscope assessment of mitotic figures, however with the development of scientific tools they were successfully replaced by more objective immunohistochemical stains with the use of universal Ki-67 evaluation. In addition, modern immunohistochemistry allows to evaluate the proliferating activity of cells much more precisely. Its importance is also confirmed in some entities in which proliferation rate assessment is not included in routine protocols [20]. Regarding the obtained results the crossing of that basic cancer mechanisms and c-jun expression pathways what stay in compliance with the literature may be suggested [19].

Very characteristic feature of neuroblastoma group is its heterogeneity what have an influence on prognosis and treatment that have to be adjusted personally to a patient. The knowledge about all of the prognostic factors that are associated with the development of neuroblastoma are crucial for assessing the patient's survival. Numerous prognostic parameters in NBL underline complexity of this disease and unknown pathways of NBL biology [21, 22].

TGFβ is produced by both immune and non-immune cells and it has a very important role in functioning of many biological processes such as embryogenesis, immunological processes and carcinogenesis [23]. TGFβ is believed to be involved in tumorigenesis and invasiveness by being a contributor in oxidative stress that is associated with cancer development [15].

Neuroblastoma is a common childhood neoplasm and the death in the patients with this disease is caused mostly by the development of metastasis. TGFβ level was proved to be increased in neuroblastoma cases and staying in connection with the patient's prognosis [24]. TGFβ as a component of ERK1/2 and TGFβ1 signaling pathways was analyzed in the patients with neuroblastoma as the potential regulator of proliferation, migration and invasion of human neuroblastoma cells. It was considered as a crucial factor in the process of cancer development and a potential therapeutic target [25].

It was revealed in presented studies that TGFβ, what stays in compliance with literature, is well known and powerful inhibitor of cell proliferation and a potent inducer of differentiation. Resistance to its action characterized many malignancies and has been attributed to alterations of its pathways and receptors, either [26]. Transforming growth factor (TGF)-β level was indeed elevated in neuroblastoma cases and found to be related to a pathway important for promoting neuroblastoma invasion and tumor metastases has been shown to be associated with the epithelial-mesenchymal transition [27, 28].

The expression of TGFβ1 was evaluated in 51 primary tumors and 17 invasion/metastasis in Wilms' tumor cells. The higher level of TGFβ1 in primary Wilms' tumor was correlated with tumor invasion and disease progression and could be a potential prognostic and predictive factor [11]. Wilms' tumor belongs to group of embryonal tumors in which development nephrogenic rests have a crucial function. TGFβ plays a critical role in embryo development and is strictly associated with cellular proliferation and its growth. It is evidenced that TGFβ has the influence on Wilms' tumor development but the correlation between them is still not fully explained.

It was evidenced that overexpression of TGFβ1 was associated with stage of the disease and presence of invasion and metastasis [29]. The same the expression of TGFβ was checked in medulloblastoma by immunohistochemistry methods in order to be associated with its metastasis and patient's survival [30].

Final study finding which is the relation of TGF-beta expression in NBL to tumor histology needs reflection on proliferation rate involvement similarly to presented glioblastoma results. Apoptotic-mitotic index included in division into favorable and unfavorable histology of NBL probably influence the obtained results.

Concluding remarks. No matter what is the way of estimation, mitotic activity is a very good indicator of the proliferation potential which reflects fundamental cancer mechanism as well as it is crossed with newly discovered pathways what regarding the obtained results found in both the examined models may be a universal finding in cancer. Although integrated science offers the most advanced approaches, it is important when drill down for molecular or even atomic biomarkers to consider the old established prognostic parameters in prognostication in individual, especially doubtful cases [21, 31].

References

1. Louis DN, Perry A, Reifenberger G et al. The 2016 World Health Organization Classification of Tumors of the Central Nervous System: a summary. Acta Neuropathol 2016; 131: 803–820.

2. Christians A, Adel-Horowski A, Banan R et al. The prognostic role of IDH mutations in homogeneously treated patients with anaplastic astrocytomas and glioblastomas. Acta Neuropathol Commun 2019; 7: 156.

3. Saadeh FS, Mahfouz R, Assi HI. EGFR as a clinical marker in glioblastomas and other gliomas. Int J Biol Markers 2018; 33: 22–32.

4. Wick W, Weller M, van den Bent M et al. MGMT testing – the challenges for biomarker-based glioma treatment. Nat Rev Neurol 2014; 10: 372–385.

5. Birkó Z, Nagy B, Klekner Á, Virga J. Novel Molecular Markers in Glioblastoma – Benefits of Liquid Biopsy. IJMS 2020; 21: 7522.

6. Taran K, Kobos J, Sitkiewicz A. Estimation of prognostic value of CD44 expression in neuroblastic tumours in children. Folia Neuropathologica 2007; 45: 126–132.

7. Schummer P, Kuphal S, Vardimon L, Bosserhoff AK, Kappelmann M. Specific c-Jun target genes in malignant melanoma. Cancer Biology & Therapy 2016; 17: 486–497.

8. Tsiambas E, Mastronikolis N, Fotiades PP, Kyrodi E. c-Jun/c-Fos complex in laryngeal squamous cell carcinoma 2020; 25: 618–620.

9. Tanyildiz HG, Kaygusuz G, Unal E, Tacyildiz N, Dincaslan H, Yavuz G. The prognostic importance of TGF-β, TGF-β receptor, and fascin in

childhood solid tumors. Pediatric Hematology and Oncology 2017; 34: 238–253.

10. Dai L, Gast A, Horska A et al. A case-control study of childhood acute lymphoblastic leukaemia and polymorphisms in the TGF-β and receptor genes. Pediatr Blood Cancer 2009; 52: 819–823.

11. Zhang L, Liu W, Qin Y, Wu R. Expression of TGF-β1 in Wilms' tumor was associated with invasiveness and disease progression. J Pediatr Urol 2014; 10: 962–968.

12. Bossy-Wetzel E. Induction of apoptosis by the transcription factor c--Jun. The EMBO Journal 1997; 16: 1695–709.

13. Raivich G. c-Jun expression, activation and function in neural cell death, inflammation and repair. Journal of Neurochemistry 2008: 107(4): 898–906.

14. Thakur A, Wang X, Siedlak SL, Perry G, Smith MA, Zhu X. c-Jun phosphorylation in Alzheimer disease. J Neurosci Res 2007; 85: 1668–1673.

15. Ramundo V, Giribaldi G, Aldieri E. Transforming Growth Factor-β and Oxidative Stress in Cancer: A Crosstalk in Driving Tumor Transformation. Cancers (Basel) 2021; 13: 3093.

16. Huang L, Quan X, Liu Z et al. c-Jun gene-modified Schwann cells: upregulating multiple neurotrophic factors and promoting neurite outgrowth. Tissue Eng Part A 2015; 21: 1409–1421.

17. Xiang D-M, Sun W, Zhou T et al. Oncofetal HLF transactivates c-Jun to promote hepatocellular carcinoma development and sorafenib resistance. Gut 2019; 68: 1858–1871.

18. Blau L, Knirsh R, Ben-Dror I et al. Aberrant expression of c-Jun in glioblastoma by internal ribosome entry site (IRES)-mediated translational activation. Proceedings of the National Academy of Sciences 2012; 109: E2875–2884.

19. Huang Z, Xia Y, Hu K, et al. Histone deacetylase 6 promotes growth of glioblastoma through the MKK7/JNK/c-Jun signaling pathway. J Neurochem 2020; 152: 221–234.

20. Taran K, Kobos J, Sporny S. Number of mitotic figures in microscopic picture of nephroblastoma histologic types-estimation of the value of the oldest known prognostic factor. Pol J Pathol 2008; 59: 43–47.

21. Coughlan D, Gianferante M, Lynch CF, Stevens JL, Harlan LC. Treatment and survival of childhood neuroblastoma: Evidence from a population-based study in the United States. Pediatr Hematol Oncol 2017; 34: 320–330.

22. Blessing MM, Alexandrescu S. Embryonal Tumors of the Central Nervous System: An Update. Surg Pathol Clin 2020; 13: 235–247.

23. Zhao H, Wei J, Sun J. Roles of TGF-β signaling pathway in tumor microenvirionment and cancer therapy. Int Immunopharmacol 2020; 89: 107101.

24. Shao J-B, Gao Z-M, Huang W-Y, Lu Z-B. The mechanism of epithelial--mesenchymal transition induced by TGF-β1 in neuroblastoma cells. International Journal of Oncology 2017; 50: 1623–1633.

25. Chen Z, Gao J, Sun J, Wu Z, Wang B. Aminoacylase 1 (ACY-1) Mediates the Proliferation and Migration of Neuroblastoma Cells in Humans Through the ERK/Transforming Growth Factor β (TGF-β) Signaling Pathways. Med Sci Monit 2021; 27: e928813.

26. Lolascon A, Giordani L, Borriello A et al. Reduced expression of transforming growth factor-beta receptor type III in high stage neuroblastomas. Br J Cancer 2000; 82: 1171–1176.

27. Micalizzi DS, Farabaugh SM, Ford HL. Epithelial-Mesenchymal Transition in Cancer: Parallels Between Normal Development and Tumor Progression. J Mammary Gland Biol Neoplasia 2010; 15: 117–134.

28. Zhen Z, Guo X, Liao R, Yang K, Ye L, You Z. Involvement of IL-10 and TGF-β in HLA-E-mediated neuroblastoma migration and invasion. Oncotarget 2016; 7: 44340–44349.

29. Shi Q, Wu H, Li Y, et al. Inhibition of Wilms' Tumor Proliferation and Invasion by Blocking TGF-β Receptor I in the TGF-β/Smad Signaling Pathway. BioMed Research International 2020; 2020: 1–10.

30. Aref D, Moffatt CJ, Agnihotri S et al. Canonical TGF-β Pathway Activity Is a Predictor of SHH-Driven Medulloblastoma Survival and Delineates Putative Precursors in Cerebellar Development: TGF-β Activity Is Prognostic in Medulloblastoma. Brain Pathology 2013; 23: 178–191.

31. Taran K, Frączek T, Sikora-Szubert A et al. The first investigation of Wilms' tumour atomic structure-nitrogen and carbon isotopic composition as a novel biomarker for the most individual approach in cancer disease. Oncotarget 2016; 7: 76726–76734.

Journal of Health Study and Medicine
2021, No. 2
pp. 25-41
DOI 10.36145/JHSM2021.08

Human Resource Management in the Group of Doctors and Nurses Employed in the Tomaszów Health Centre in Tomaszów Mazowiecki

Marta Świerczyńska[1]
https://orcid.org/0000-0003-0316-1953

Monika Zgoła[1]
https://orcid.org/0000-0003-0852-6616

Małgorzata Widawska[2]
https://orcid.org/0000-0003-3338-6029

Paulina Stasiołek[1]
https://orcid.org/0000-0003-1300-1187

Anna Gawron-Skarbek[3]
https://orcid.org/0000-0002-2953-4060

[1] Social Medicine Department, Medical University of Lodz, Poland
[2] District Sanitary and Epidemiological Station in Radomsko, Poland
[3] Department of Hygiene and Health Promotion, Medical University of Lodz, Poland

Address for correspondence

Marta Świerczyńska
Social Medicine Department, Medical University of Lodz
7/9 Żeligowskiego St., 90-752 Lodz, Poland
knopmarta@wp.pl

Abstract

Introduction: *Human resource management is a key process in organisation management. Skilful management of the staff, setting the goals and motivating the employees to high-level performance of their tasks help to achieve the organisation's goals consistently.*

Purposes: *The study was aimed to analyse the process of motivating the doctors and nurses working in the Tomaszów Health Centre and to investigate the staff's satisfaction with the incentives used in their workplace.*

Materials and methods: *The study population covered fifty doctors and fifty nurses employed at four departments of the Tomaszów Health Centre. The study involved an analysis of the factors which motivate and demotivate the THC staff and an investigation of the incentives used towards the staff. The level of the study population (doctors and nurses) satisfaction with the methods of their motivation was also examined.*

Results: *The analysis of the results revealed that the most desired factors with the most positive impact on the staff motivation level include appreciation bonuses (40% of the respondents), good atmosphere at work (38%) and the possibility to enhance one's qualifications (21%). The stimulus which affects the effectiveness of the staff's performance of their duties is an orally expressed praise of the CEO/president, boss or superior (31%). The doctors and nurses in the study population do not feel satisfied with the incentive scheme used in the THC (69%).*

Conclusions: *In-cash factors play the most important role in the incentive scheme for the majority of THC's employees. Most of the doctors and nurses working for THC do not feel satisfied with the incentives used in their workplace.*

Key words: *motivation, human resource management, incentive schemes*

Since the 1990s, the health care system in Poland has been evolving dynamically. The health care sector has become a powerful branch of Poland's economy owing to the implementation of public health insurance, Health Care Fund and National Health Fund as well as further transformations which started when Poland joined the European Union.

The management of health care centres gained a new dimension, and the centres were supposed to become efficiently operating businesses.

One should remember that the system is not closed. It is dynamically affected by politics and economy. "The health care system does not function in a political, economic or institutional vacuum. Its structure and functions are determined by various legislative acts, starting from the Constitution through to ratified international agreements, regulations, directives and decisions of the European Union as well as acts and statutory instruments, regulations and local laws, which include the regulations issued by the President of the NHF" [1].

The management of human resources is among the key management areas of a health care organisation. Human resources are the organisation's asset which contributes to the implementation of its objectives as a company, once managed skilfully and timely [2].

According to such authors as M. Armstrong, A. Pocztowski, T. Listwan and A.R. Griffin the management of human resources can express the contemporary personnel function of an organisation, emphasising some its characteristics, including but not limited to:

- approaching an employee as the source of the organisation's key asset, emphasising the implementation of an individual's specific objective,
- conceptual linking of HR matters with the economic strategy,
- management activation,
- instigating measures aimed to combine the employer's and employees' interests [3].

According to the above, management can be described as the determination of future objectives and adequate allocation of the organisation resources, ensuring an increase in the unit's employees value, including

the available knowledge and information resources, as well as more effective solutions to specific issues [4].

Human resource management shall be a planned action implementing the organisation's objectives and tasks as well as taking into account the staff's needs and development [5]. The impact on the employees has to be planned for the objectives – it develops the human resources by affecting the number of people in the staff groups; matches the staff's quality features based on their involvement and the results achieved owing to the quality and effectiveness of fulfilling their duties [6]. The execution of the function related to human resources covers the processes of the staff acquisition to satisfy the organisation's needs, adapting the number of the employees and their employment basis to the intended objectives, future staff development and making them redundant, if necessary. As part of the human resource management function, the staff carries out activities bearing in mind the abovementioned assumptions. The activities can be divided into two groups: regulatory, which include HR planning and motivating, and material, which include training and staff selection. Any decisions that are made by the management staff should be of strategic importance for the whole organisation [7].

The human resource management actors include persons who directly participate in the execution of the organisation's HR function. The actors are divided into internal and external ones [8]. The internal actors are the organisation owners and proxies, managers of the particular organisational unit, HR team, human resources, and their representatives (trade unions) [9]. Internal actors are responsible for HR management in the organisation and implement measures aimed to develop human resources [10]. The role of the external actors, such as legislative bodies, national authority bodies, supervision institutions (e.g., National Labour Inspectorate) or control institutions (e.g., Supreme Audit Office) is to develop the conditions of the organisation's human resource management execution by laws [11].

Efficient management of the organisation's human resources by the management bodies is based on logical guidelines in the form of human

resource management strategic models. The reference publications mention two basic models of human resource management: Michigan and the Harvard model [12]. The distinction was made in the USA in 1984.

The Michigan model, which is a hard, push or strict human resource management model, approaches the HR function as a concept of purely strategic management of human resources. In the model developed by a group of researchers from the University of Michigan, the organisation's strategy plays the supreme role. The management staff develops the organisational structure and manages the human resources according to the strategy. The approach to management in the Michigan model is characterised by a vision of a human – employee being a resource. That is why the model focuses on the benefits for the organisation and the costs related to human resources [13]. In this model, the human resources are managed in the same way as the organisation's other resources. The functions of such management include staff selection, evaluation of the staff's work outcomes, staff rewarding and their development. The functions matter on all management levels, including strategic, tactical and organisational level, and in combination they form a system based on the aforementioned concept.

The Harvard model, in turn, called a soft or pull model, focuses on communication, teamwork and the employee's individual determinants and strengths. In this approach, it is the creative energy of the organisation and the source of its competitiveness [14]. Humans are the Centre of attention, along with the impact they may exert on the organisation. The management offers more opportunities for the staff and welcomes proposals of new solutions. The manager's role focuses on working with the staff, motivating them, emphasising good practices, strengthening engagement, appreciation for self-reliance, initiative and creativity [15]. The Harvard model differentiates the following areas as management functions: staff participation; selection, relocation and dismissal of the human resources; employee incentive scheme and organisation of their work [16].

The management staff takes advantage of both abovementioned models in human resource management. Some managers prefer to use only

one of the models. Both approaches have their advantages and disadvantages, and both greatly affected the theory of organisation operation and management as well as the practice of human resource management. They gave grounds for subsequent research in the area.

Motivation is among the essential functions of human resource management [17]. An efficient manager will skilfully use staff motivation methods to achieve the organisation's objectives [18]. Adequate motivation, which is a set of factors which stimulate and support the staff's activity, makes the employees achieve their objectives faster and more efficiently. It is a very important growth factor of the staff effectiveness, and consequently the factor which increases the organisation's competitiveness [19]. The manager shall approach their employees individually, diagnose their individual needs and expectations, develop adequate working conditions and select the management methods that will help the organisation fulfil its objectives and see measurable results of the employee's working outcomes [20]. There is a plethora of incentives available. From the formal point of view, motivation can be divided into internal (attribute-based) and external (function-based). In the first case, the motivation results from internal processes which affect the initiation, orientation and supporting of actions and behaviours aimed to achieve the objectives. The other type of motivation (external) applies to conscious and intentional influence on the employees' behaviour by using external factors.

The literature differentiates between two types of employee motivation: material and non-material. The first one involves "development of financial conditions that enable the staff acquisition and retention and their engagement in the fulfilment of the organisation's objectives" [21]. In addition to remuneration (salary) it applies to appreciation bonuses, in-cash prizes and in-kind prizes. Nowadays organisations try to outdo one another in their ideas to attract the most valuable employees on the market to work for them and to effectively realise the company's objectives, by offering such incentives as life insurance or other fringe benefits, including fitness passes, food vouchers etc. [22].

Non-material motivation is based on offering conditions for professional development, flexible working hours, creating a good atmosphere at work and trusting the employees [23]. Non-material incentives have currently been taken more into consideration when choosing a place to work. It is an important factor which affects working effectiveness [24].

It is the management staff's role to select the types of incentives for their staff that help to achieve the organisation's objectives most effectively [25]. The ability to motivate their subordinates is by far among the most required features of a manager. "It is important to learn what factors motivate the staff to greater working effectiveness, involvement in the organisation's issues or bring satisfaction with the work done" [26].

Purpose

The purpose of the study was to analyse the primary functions of human resource management and motivating the group of doctors and nurses working for the Tomaszów Health Centre in Tomaszów Mazowiecki. The study was aimed at analysing the factors which motivate the staff working in the Centre, the incentives used and the non-satisfied needs of the doctors and nurses.

Material and methods

A survey based on original and anonymous questionnaires addressed to the staff of the Tomaszów Health Centre was carried out in the Centre in 2018.

The study population included fifty doctors and fifty nurses working in four departments: Department of General and Oncological Surgery, Department of Internal Medicine, Department of Neurology and Department of Paediatrics.

75% of the study population were females, and 25% were males. The analysis of the study material revealed (Tables 1 and 2) that over half of the study population were employees over 40 years of age. The majority of the staff were people with long job seniority.

Table 1. The age structure of the THC employees' group participating in the survey

Age of the survey participants	Number of persons [N = 100]
20–29 years	29
30–39 years	6
40–49 years	58
50–59 years	6
Over 60	1

Source: own study.

Table 2. The job seniority structure of the THC employees participating in the survey

Job seniority	Number of persons [N = 100]
Up to 5 years	14
Up to 10 years	12
Up to 15 years	22
Up to 20 years	30
Up to 30 years	20
Over 30 years	2

Source: own study.

Analysis of the study results

According to the survey (Table 3) material factors are most motivating for over half of the study population (53%). The most popular material factors among the employees included an appreciation bonus (40%) and remuneration as such (24%). The responses concerning in-cash prizes, evening rates and pay rise were given by 13%, 12% and 11% of the study population.

The study revealed that 69% of the employees participating in the survey were not satisfied with their salary.

The study population was also examined for non-salary factors (Table 4). The degree of their influence on the motivation of the Tomaszów Health Centre's staff was investigated.

Table 3. Material factors affecting the staff motivation in the THC

Material incentive	Number of persons [N = 100]
Appreciation bonus	40
Salary	24
In-cash prizes	13
Pay rise	11
Evening rates	12

Source: own study.

Table 4. Non-salary factors motivating the staff for work

Non-salary factors affecting the motivation	Number of persons [N = 100]
Good atmosphere at work	38
Training/learning at the employer's expense	21
Praises and distinctions	21
Holiday subsidies	11
Reimbursement of the costs of commuting to/from work	6
Only money motivates me	3

Source: own study.

According to the study, a good atmosphere at work was motivating for 38% of the study population. 21% of the respondents are motivated by a training scheme, and any forms of education at the employer's expense. Praises and distinctions stimulate the equivalent number of people. 11% indicated holiday subsidies and 6% reimbursement of the costs of commuting as the incentives which motivate them for work.

3% of the respondents declared that the financial factor is the only aspect that affects their motivation level.

The THC staff were also asked about the most effective non-material incentives in their work (Table 5).

Praise expressed orally by the superior and addressed directly to the employee turned out to be the most substantial incentive of all (31%).

Other motivating factors included autonomy and freedom of action (22%), praise expressed in public (16%), flexible working hours (12%), certificates of appreciation (10%) and the superior's trust (9%).

Table 5. Non-material incentives which are most motivating for work

In-kind incentives as the most motivating factors	Number of persons [N = 100]
Trust to the employer/superior	9
Individual praise expressed by the superior orally	31
Praise expressed in public	16
Autonomy and freedom of action	22
Flexible working hours	12
Certificates of appreciation	10

Source: own study.

The motivating person plays a vital role in the process of motivating the staff [24]. The study carried out in the Tomaszów Health Centre revealed that the persons who motivate doctors and nurses most include the persons of higher seniority in the organisation (35%), superiors (27%) and workmates (24%). Friends and relatives (10%) and patients (4%) were selected as the least motivating for work (Figure 1).

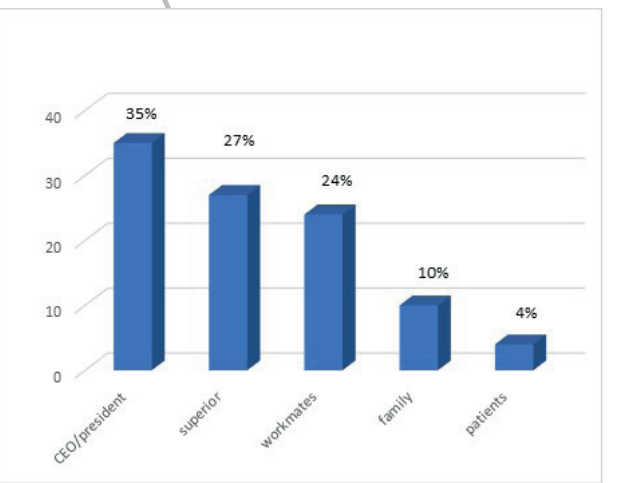

Figure 1.

The study carried out in 2018 was also aimed at investigating the in--cash and in-kind factors that reduce the staff's motivation in the THC in Tomaszów Mazowiecki to work. The questionnaire included multiple-choice questions. A lack of bonuses/in-cash prizes or their granting always to the same persons turned out to be the most demotivating in--cash factors (89%). The respondents indicated the following factors as those which reduce their motivation to work: a lack of pay rises (86%), a lack of bonuses or a low value of the bonus (79%) (Figure 2).

Figure 2.

The in-kind factors regarded by the THC staff as most reducing the motivation to work included bad atmosphere at work – as many as 90 persons pointed out this factor as important. A lack of training or development opportunities was the second most common answer selected by the employees (88 of the respondents) as demotivating them for work. The staff motivation was also reduced by a lack of holiday subsidies (66% of the respondents) and working only part-time with no possibility to be employed on a full-time basis.

The survey revealed that slightly more than half of the doctors and nurses in the study population (55%) were improving their professional skills in the last five years. 45% of the respondents did not develop their professional skills in the reference period.

According to the survey participants, the THC's staff are mainly motivated by in-kind incentives. When asked about the type of incentives

used in their workplace, 57% of the respondents indicated in-kind incentives as dominating. 29% of the study participants answered that these were mainly in-cash incentives, while 14% mentioned both types of incentives.

The final part of the survey applied to the THC staff's satisfaction in reference to their sense of being motivated by their superiors. As much as 68% of the doctors and nurses participating in the study gave negative answers – they did not feel supported by their superior's motivation.

The last question in the survey carried out in the THC referred to the non-satisfied needs of the staff (Diagram 3).

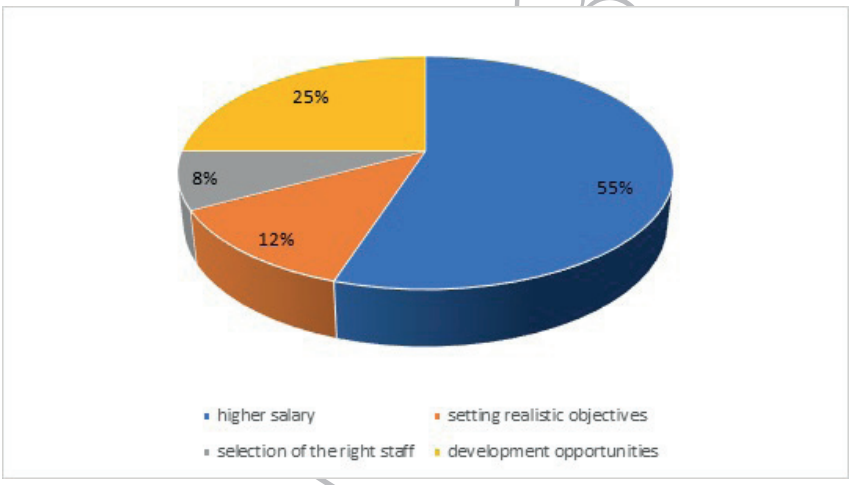

Diagram 3.

Over half of the respondents (55%) said their employer should give them a pay rise. 25% of the respondents believed that the staff's motivation would increase, if the employer offered conditions for development, by offering training and courses; 12% of the study participants answered that setting realistic objectives could improve motivation to work. Selecting the right staff was the answer given by 8% of the respondents.

Conclusions

1. Over half of the THC's staff participating in the study were motivated by material incentives.
2. An appreciation bonus is the most critical in-cash factor for 40% of the respondents.
3. 69% of the doctors and nurses participating in the study were not satisfied with their remuneration level.
4. In-kind incentives that most affect the study population's motivation include good atmosphere at work and the opportunity to improve one's skills by participating in courses and training.
5. According to 35% of the respondents, the boss or manager is the person whose praises are most motivating.
6. The factors which demotivate the THC's staff most include a lack of rewards or their limited scope, as well as the fact that the rewards are granted to the same employees every time.
7. 90% of the doctors and nurses participating in the study admitted that bad atmosphere at work demotivates them.
8. According to the respondents, in-kind incentives are the primary type of incentives used in the THC.
9. As much as 68% of the study population do not feel motivated to work.

References

1. Nojszewska E. System Ochrony Zdrowia w Polsce. Warszawa: Wolters Kluwer; 2011, p. 49.

2. Sidor-Rządkowska M. Zarządzanie zasobami ludzkimi w administracji publicznej. Warszawa: Wolters Kluwer; 2013, p. 43.

3. Koryakovtseva OA, Doronina J, Panchenko T, Karabulatova IS, Abdullina ZM. Research of Category "Motivation" as a Basic Tool of Personnel Management. International Review of Management and Marketing 2016; 6(S1): 293–299.

4. Kęsy M. Kształtowanie zdolności menedżerskich personelu medycznego w szpitalach. Kraków: Wydawnictwo UJ; 2013, p. 69.

5. Stabryła A, Wawak S. Metody badania i modele rozwoju organizacji. Kraków: Mfiles.pl; 2012, p. 83.

6. Glišović M. A, Jerotijević G, Jerotijević Z. Modern approaches to employee motivation. Ekonomika 2019; 65(2): 121–133 [Internet]. Available from: https://www.researchgate.net/publication/334310227_Modern_approaches_to_employee_motivation [cited April 2019].

7. Marciniak J, Meritum HR. Warszawa: Wolters Kluwer; 2013, p. 48.

8. Zapłata S. Zarządzanie jakością w przedsiębiorstwie. Warszawa: Wolters Kluwer; 2009, p. 130.

9. McGregor L, Doshi N. How company culture shapes employee motivation. Harvard Business Review 2015 [Internet]. Available from: https://hbr.org/2015/11/how-company-culture-shapes-employee-motivation [cited 25.11. 2019].

10. Harter J, Askins A. What great managers do to engage employees. Harvard Business Review [Internet]. Available from: https://hbr.org/2015/04/what-great-managers-do-to-engage-employees [April 2019].

11. Tracy B. Learning leadership: Eight key skills that make an effective manager. Forbes [Internet]. Available from: https://www.forbes.com/sites/forbescoachescouncil/2017/03/13/learning-leadership-eight-key-skills-that-makean-effective-manager/#2e22bf621354 [cited 13.03.2019].

12. Jamka B. Czynnik ludzki we współczesnym przedsiębiorstwie – zasób czy kapitał? Warszawa: JAK; 2011, p. 110.

13. Huseyn Bilgin M, Danis H, Demir E, Ugur C. Country experiences in Economic Development, Management and Entrepreneurship. Proceedings of the 17th Eurasia Business and Economics Society Conference. Cham: Springer; 2018.

14. Jensen JD. Employee Motivation: A Leadership Imperative. International Journal of Business Administration 2018; 9(2): 93–98.

15. Zastawna A. Zarządzanie pracownikami – twarda gra w miękkim stylu. BiznesPlus 30 May 2018 [Internet]. Available from: http://www.biznesplus.pl/zarzadzanie-pracownikami/.

16. Pocztowski A. Zarządzanie zasobami ludzkimi. Zarys problematyki i metod. Kraków: Antykwa; 1998.

17. Zając S. Wpływ motywowania pracowników na jakość i efektywność pracy w przedsiębiorstwie. Prace Naukowo-Dydaktyczne PWSZ im. J. Pigonia w Krośnie 2014; 67: 185–202.

18. Baergie S. The Science of Motivation [Internet]. Available from: https://hrmagazine.co.uk/article-details/the-science-of-motivation [cited 15.10.2018].

19. Badubi RM. Theories of Motivation and Their Application in Organisations: A Risk Analysis. International Journal of Innovation and Economic Development 2017; 3: 44–51.

20. Aremu I. The key to economy recovery is motivated and skilled workforce. Daily Trust [Internet]. Available from: https://www.dailytrust.com.ng/news/labour/-the-key-to-economyrecovery-is-motivated-and-skilled-workforce/189972.html [cited 01.06.2019].

21. Listwan T. Zarządzanie kadrami. Warszawa: C.H. Beck; 2002.

22. Karna WJ, Knap-Stefaniuk A. Motywowanie Pracowników jako wyzwanie w zarządzaniu zasobami ludzkimi. Studia i Prace WNEIZ US 2017; 48(2): 162–171.

23. Essays, UK. Literature Review of Theories of Motivation in Employees [Internet]. Available from: https://www.ukessays.com/essays/business/literature-review-of-theories-of-motivation-in-employees-business-essay.php?vref=1 [cited November 2018].

24. Beltrán-Martin I, Bou-Llusar JC. Examining the intermediate role of employee abilities, motivation and opportunities to participate in the relationship between HR bundles and employee performance. BRQ Business Research Quarterly 2018; 21(1): 99–110.

25. Kłobukowska K. Materialne i niematerialne narzędzia motywowania pracowników [Internet]. Available from: http://goodpoint.blog/materialne-niematerialne-narzedzia-motywowania-pracownikow [cited December 2018].

26. Haider M, Aamir A, Abdulhamid A, Hashim M. Literature Analysis on the Importance of Nonfinancial Rewards for Employees' Job Satisfaction. Abasyn Journal of Social Sciences 2015; 8(2): 341–354.

Journal of Health Study and Medicine
2021, No. 2
pp. 43-60
DOI 10.36145/JHSM2021.09

Smoking Tobacco – a Risk Factor for Breast Cancer

Marta Szkiela[1]
https://orcid.org/0000-0003-3631-5082

Ewa Kusidel[2]
https://orcid.org/0000-0002-0138-6991

Ewa Siwińska-Beck[3]

Teresa Makowiec-Dąbrowska[4]
https://orcid.org/0000-0003-0373-9073

[1] Department of Hygiene and Epidemiology, Faculty of Health Sciences, Medical University of Lodz, Poland
[2] Department of Spatial Econometrics, Faculty of Economics and Sociology, University of Lodz, Poland
[3] Department of Hygiene and Health Promotion, Faculty of Health Sciences, Medical University of Lodz, Poland
[4] Department of Work Physiology and Ergonomics, Nofer Institute of Occupational Medicine in Lodz, Poland

Address for correspondence

Marta Szkiela
Department of Hygiene and Epidemiology, Faculty of Health Sciences
Medical University of Lodz
Żeligowskiego 7/9 St., Lodz 90-752, Lodz, Poland
marta.szkiela@umed.lodz.pl

Abstract

Introduction: *In 2009, the International Agency for Research on Cancer (IARC) stated that there was "limited evidence of a cause and effect relationship between an increased risk of breast cancer and smoking". Environmental tobacco smoke is known to cause cancer by exerting both carcinogenic and genotoxic effects. The aim of the study was to assess the relationship between smoking (active and passive) and breast cancer risk.*

Material and methods: *The case/control study was conducted from 2015––2019 in the Lodz region. The case group included 384 women with breast cancer; the control group included 298 healthy women. The research tool was an anonymous questionnaire.*

Results: *In the group of 682 respondents, 63% have ever smoked cigarettes (65.4%/60.1% in the case/control group respectively), 22.1% were current smokers, 67% were exposed to passive smoking. In the majority of cases, women were exposed to smoking by the husband (86%), the father (55.5%) or both (43.2%). Breast cancer risk is 253–271% higher for smokers (current – past) in comparison to the group of non-smokers, never exposed to passive smoking. The risk for passive smokers is 139% higher than for those not exposed.*

Conclusions: *Exposure to environmental tobacco smoke increases the risk of breast cancer. Public awareness of the negative consequences of passive smoking should be raised.*

Key words: *tobacco smoking, passive smoking, breast cancer, tobacco-related diseases, breast cancer risk factor*

Introduction

The problem of cigarette smoking (active and passive) severely affects the entire world's population. According to data on prevalence of smoking in Europe (collected in the European Health Interview Survey (EHIS)), almost one in four EU28 inhabitants smoke cigarettes (23.9% to be exact) and almost one in five women (19.5%). However, smoking varies widely from country to country – from Bulgaria, Greece, and Turkey, where more than 30% of the population smokes, to Sweden, where the percentage of smokers is 16.7%. Regarding the percentage of smokers by sex, for all EU28 countries, the percentage of smokers is higher for men than for women, with the highest difference being Lithuania (40.3/12.3% smoking men/women) and the lowest Sweden (17.4/16.0%). For Poland, the percentage of smoking men is higher than women (32.5/20.3%) [1, 2]. Kaleta research [3] shows that among professionally active people the percentage of smokers is higher (daily smoking was declared by 37.3% of men and 28.2% of women).

According to the World Health Organization (WHO), smoking kills approximately 8 million people a year (including 1.2 million deaths attributed to second-hand tobacco smoke [SHS]) [4]. The percentage of people who die of tobacco-related diseases is higher than the sum of victims of tuberculosis, malaria and HIV/AIDS. Among the smoking-related causes of deaths, the list is topped by cardiovascular diseases, respiratory failure in the course of chronic obstructive pulmonary disease (COPD), and malignant cancer, smoking is also an important risk factor for stroke, blindness, deafness, back pain, osteoporosis, and peripheral vascular disease [5]. As early as 1964, the carcinogenic properties of tobacco smoke were identified, especially those of several polycyclic aromatic hydrocarbons which featured in the Surgeon General's report on smoking and health [6]. In 2009, the International Agency for Research on Cancer (IARC) stated that there was "limited evidence of a cause and effect relationship between an increased risk of breast cancer and smoking" [7].

Breast cancer is the most frequently diagnosed cancer among women (24.2% of all cancers) in a majority (140/184) of countries worldwide, accounting for one quarter of the total. It is also a leading cause of cancer--related deaths in women (15.0%) [8]. In Poland, in 2018, breast cancer was the most common cancer among women (22.5% of all cancers) and the second (after lung cancer) cause of death due to malignant tumors (15.0% of all cancer-related deaths) [9].

The aim of the study was to assess the association between active and passive smoking and breast cancer, controlling for most of the above-mentioned factors of this disease and to establish prognostic implications if any.

Material and methods

The case/control study was conducted from 2015–2019 in the Lodz region. The case group included 494 women diagnosed with malignant breast cancer. The women from the case group were patients of the Oncological Surgery Department and the 2nd Department of Oncological Surgery, Oncological Surgery Clinics of the Provincial Specialist Hospital M. Kopernik in Łódź; the surgery department of Poddębice Health Center; and the Provincial Specialist Hospital M. Skłodowska-Curie in Zgierz. The control group included 515 women without a diagnosis of breast cancer. A detailed description of the study design and study population, including the selection of women for the case/control groups and a description of the research tool, can be found in our previous article: "Night Shift Work-A Risk Factor for Breast Cancer" [10]. The research tool was an anonymous and voluntary questionnaire.

Respondents were asked: "Have you ever smoked cigarettes for more than six months?". If they replied positively, they were asked about their smoking status, with possible variants of responses: yes or no. For "yes" answers a further distinction was made: "former smoker" and "active smoker." The other question: "Have you ever lived with someone who smoked regularly at home?" was asked to identify women exposed to secondhand

smoke. For "yes" answers, there were further questions about relatives who smoked (father, mother, siblings, husband). Respondents were also asked: "How many smokers together have lived with you throughout your life?", with the answer containing the number of people [1, 2, 3, 4, 5].

Respondents also answered questions about their exposure to passive smoking ("exposed", "not exposed").

The study design received a positive opinion from the Bioethics Committee at the Medical University of Lodz (RNN/236/15/EC of 22 September 2015). The part of the survey concerning smoking was completed by 682 women.

The following statistical methods were used in the analysis: (1) t-test for mean value, (2) proportions – to calculate the percentage of a given category in the case and control groups; (2) simple and multiple logistic regression to estimate the odds ratio (OR). Statistical calculations were carried out in: (1) a Microsoft Excel spreadsheet (frequency distribution of the variables, contingency tables); (2) a Gretl econometric package (t--test and logit model selection); and (3) Statistica 13.3 (OR and CI for the final version of the logit model). Predictor reduction in the logit model was carried out using a backward elimination model selection procedure, which started with the most general model and eliminated one variable at a time until the best model was reached (i.e. when all right-side variables were statistically significant for p 0.05).

Results

In the group of 682 respondents who completed the section on smoking, 430 (63%) have ever smoked for at least for six months in their life (65.4% and 60.1% in the case/control group respectively). Up to 279 (40.9%) of the smokers successfully quit smoking (42.7%/38.6% in the case/control group). There were 151 current smokers (22.1% of the whole sample, 22.7%/21.5% in the case/control group).

An average number of smoked cigarettes is similar in the case/control group (15.2/14.7 cigarettes per day) with no significant differences in the

group of current (15.6/14.3) and former (15.0/14.9) smokers. What significantly differences case/control group is the years of smoking which is significantly higher in the case group (31.5 vs 28.6 average years of smoking). This difference is made by former smokers who have longer history of smoking (28.2 vs 24.9 years of smoking).

The whole characteristic of the sample is presented in Table 2, which shows that the most significant differences between the case and control group occur in terms of the following factors:

- BMI > 25 – in the case group the percentage of women who are obese is 104.1% (for BMI ≥ 30) and 72% (for BMI 25–30) higher than the control group;
- late menopause – in the case group, the percentage of women who had menopause at age 55+ is 88.4% higher than the control group;
- Smoking – in the case group, the percentage of women who are non-smokers not exposed to smoke is 70.2% lower than the control group;
- number of smokers with whom the respondent lived – in the case group, the percentage of women who live at home with two smoking persons is 76.1% greater than the control group.

The characteristics from Table 1 and 2 show that history of smoking in the group of current and past smokers is fairly similar. Confirmation of this can be found in the table below, where the ORs for the current and past smokers differ little, and are even slightly higher for former smokers (this is due to the longer history of smoking in the case group of former smokers then in control group of former smokers – see last row of Table 1).

The OR for smokers in contrast to the group of non-smoking women is similar for current and past smokers and equal to 1.22–1.28 and shows that risk of breast cancer is 22–28% higher in the group of smokers and past smokers, but is statistically insignificant. We observed high percentage (67%) of respondents exposed to passive smoking (456 out of 682). In the majority of cases, women were exposed to smoking by the husband (86% of all exposed to passive smoking women) or father (55.5%) or both (43.2%) – see Table 2.

Table 1. Smoking status nn case and control group

	Case			Control			p-value for case/control average' equality
	N	average	stan. dev.	N	average	stan. dev.	
	Age						
Smokers	251	62.8	9.8	179	61.5	11.3	0.204
• current	87	60.4	8.4	64	59.2	9.6	0.416
• former	164	64.1	10.3	115	62.8	12	0.333
Non-smokers	133	62.3	12.4	119	60.5	9.2	0.196
	Number of cigarettes						
Smokers	251	15.2	7.0	179	14.7	5.8	0.434
• current	87	15.6	7.9	64	14.3	5.3	0.256
• former	164	15.0	6.5	115	14.9	6.1	0.897
	Years of smoking						
Smokers	251	31.5	12.6	179	28.6	13.9	0.025
• current	87	36.4	9.9	64	35.0	10.5	0.404
• former	164	28.2	12.7	115	24.9	14.2	0.043

Source: own calculations based on case-control data.

Table 2. Characteristics of the cases and control groups

Categories	Case	Control	Case	Control	Case/Control % difference
	N = 384	N = 298	%	%	
Smoking					
Smoker	87	64	22.7	21.5	5.6%
Former smoker	164	115	42.7	38.6	10.6%
Non-smoker	133	119	34.6	39.9	-13.3%
Passive smoking—who smoked at home					
Total	289	167	75.3	56.0	34.5%
Father	168	85	43.8	28.5	53.7%
Mother	27	18	7.0	6.0	16.7%
Husband	251	141	65.4	47.3	38.3%
Father and husband	135	62	35.2	20.8	69.2%
Passive smoking – how many smokers lived with respondent					
Total	270	157	70.3	52.7	33.4%
1	126	85	32.8	28.5	15.1%
2	127	56	33.1	18.8	76.1%
≥ 3	17	16	4.4	5.4	-18.5%
Smoking including passive smoking					
Smoker exposed to smoke	60	31	15.6	10.4	50.0%
Former smoker exposed to smoke	111	56	28.9	18.8	53.7%
Non-smoker exposed to smoke	118	80	30.7	26.8	14.6%

Smoking excluding passive smoking					
Smoker not exposed to smoke	27	33	7.0	11.1	-36.9%
Former smoker not exposed to smoke	53	59	13.8	19.8	-30.3%
Non-smoker not exposed to smoke	15	39	3.9	13.1	-70.2%
Age					
35–44	17	15	4.4	5.0	-12.0%
45–54	72	62	18.8	20.8	-9.6%
55–64	136	114	35.4	38.3	-7.6%
65–74	115	83	29.9	27.9	7.2%
75+	44	24	11.5	8.1	42.0%
Degree of urbanization					
Cities	91	69	23.7	23.2	2.2%
Towns and suburbs	170	142	44.3	47.7	-7.1%
Rural areas	115	73	29.9	24.5	22.0%
No answer	8	14	2.1	4.7	
Educational level					
ISCED 0–2	51	31	13.3	10.4	27.9%
ISCED 3–4	270	197	70.3	66.1	6.4%
ISCED 5–8	62	68	16.1	22.8	-29.4%
No answer	1	2	0.3	0.7	

Marital status					
Married	260	211	67.7	70.8	-4.4%
Widow	62	33	16.1	11.1	45.0%
Divorced (in separation)	23	19	6.0	6.4	-6.3%
Never married	20	21	5.2	7.0	-25.7%
No answer	19	14	4.9	4.7	
Employment history					
III shifts worker	148	73	38.5	24.5	57.1%
worker	220	210	57.3	70.5	-18.7%
Non worker	8	10	2.1	3.4	-38.2%
No answer	8	5	2.1	1.7	
BMI					
18–25	159	193	41.4	64.8	-36.1%
25–30	144	65	37.5	21.8	72.0%
≥ 30.00	76	29	19.8	9.7	104.1%
No answer (or BMI < 18)	5	11	0.8	1.7	
Age of first menstrual period					
10–12	133	74	34.6	24.8	39.5%
13–15	208	184	54.2	61.7	-12.3%
16–18	38	39	9.9	13.1	-24.4%
No answer	5	1	1.3	0.3	

Age of menopause					
≤ 44	20	20	5.2	6.7	-22.4%
45–49	42	24	10.9	8.1	34.6%
50–54	145	139	37.8	46.6	-18.9%
≥ 55	100	41	26.0	13.8	88.4%
No answer	77	74	20.1	24.8	
Number of pregnancies					
0	44	37	11.5	12.4	-7.3%
1	81	80	21.1	26.8	-21.3%
2	145	105	37.8	35.2	7.4%
3	76	54	19.8	18.1	9.4%
≥ 4	38	22	9.9	7.4	33.8%
Sum of duration (in months) of breastfeeding					
< 6	190	104	49.5	34.9	41.8%
6–12	107	107	27.9	35.9	-22.3%
> 12	87	87	22.7	29.2	-22.3%

Source: own calculations based on case-control data.

If smokers are compared with the group of non-smoking women not exposed to passive smoking, the ORs are significantly higher (OR = 3.53–3.71). This shows that the risk of breast cancer is 253––271% higher for smokers (current–past) in comparison to the group of non-smokers who were never exposed to passive smoking. The breast cancer risk for passive smokers is 139% higher than for those not exposed (OR = 2.39). If we exclude from passive smokers current and past smokers, the OR = 3.84 and this shows the 'clear' effect of only passive smoking (non-smoking women exposed to smoke have a nearly three times higher risk of breast cancer than non-smoking women never exposed to smoke). Finally, we calculated the OR for non-smokers not exposed to smoke, OR = 0.27, which shows that this group have a 73% lower risk of breast cancer than all the others.

Below are the results of the multiple logistic regression, which includes all the others factor from Table 2 which, along with smoking, can affect breast cancer (a backward elimination procedure was used to reduce the model to a form with only statistically). Results from Table 4 confirm that non-smoking significantly lowers the odds of breast cancer (OR = 0.37; 95% CI: 0.19–0.71), but only if non-smokers were not exposed to passive smoking. Similar to the study [10], the most crucial risk factors of breast cancer are high BMI, which increases the risk by 147–211% and night shift work, which increases the risk nearly twofold. Late menopause and short (or non) period of breastfeeding are factors with an influence of 71 and 44% respectively. Differences between the ORs from Table 4 and [10] results from the different group sizes – here we analyzed only 682 persons who answered all the questions about smoking, so currently estimated OR = 0.37 more precisely measures the impact of non-smoking on breast cancer.

As non-smoking (with no exposure to passive smoke) occurs in a significant percentage of breast cancer cases, we were interested in which sociodemographic factors are favorable to this group. Logistic regression for non-smokers not exposed to smoke, considering the sociodemographic factors from Table 2, shows that only one of these factors signifi-

cantly increases the chances of being in this group. This was the higher education level (ISCED 5–8) for which there is an OR = 2.94, showing that women with a high educational level have twice higher odds of being non-smokers not exposed to smoke than women without high education.

Table 3. Impact of smoking on breast cancer (simple logistic regression).

Categories	OR	Se	95%	CI
Smokers vs non-smokers	1.22	0.21	0.81	1.83
Former smokers vs non-smokers	1.28	0.18	0.91	1.80
Smokers vs non-smokers not exposed to smoke	3.53	0.35	1.80	6.96
Former smokers vs non-smokers not exposed to smoke	3.71	0.33	1.95	7.04
Exposed to smoke vs not-exposed to smoke	2.39	0.17	1.72	3.30
Non-smokers exposed to smoke vs non-smokers not exposed to smoke	3.84	0.34	1.98	7.42
Non-smokers not exposed to smoke vs all the others	0.27	0.31	0.15	0.50

Source: own calculations in Statistica 13.3.

Table 4. Statistically significant odds ratios from multiple logistic regression

Variable	OR	Se	95% CI	
Constant	0.59	0.14	0.45	0.79
BMI >= 30	3.11	0.25	1.91	5.07
BMI 25–30	2.47	0.19	1.71	3.57
Night shift work	1.89	0.18	1.32	2.69
Age of menopause 55+	1.71	0.21	1.12	2.61
Breastfeeding <= 6	1.44	0.18	1.01	2.05
Non-smokers not exposed to smoke	0.37	0.33	0.19	0.71

Source: own calculations in Statistica 13.3.

Discussion

In most epidemiological studies the findings have indicated that both active and passive smoking can increase the risk of breast cancer. In our previous article [10], not-smoking was a significant factor which lowered the OR of breast cancer by 60% (OR = 0.40). Similar results have been

obtained in this study (OR = 0.37). The increased risk may be associated with smoking large quantities of cigarettes, smoking for a long duration, and passive smoking [11–15]. In a nationwide Danish Nurse Cohort Study on 21 867 female nurses, Andersen ZJ et al. found association between active smoking and breast cancer risk. The percentage of active smokers was higher to that observed in our study, 33.7% of nurses were current and 30.0% were former smokers. Authors found increased breast cancer risk of 27% in current (HR: 1.27; 95% CI: 1.11–1.46) smokers and 18% in ever (HR: 1.18; 95% CI: 1.04–1.34) smokers. The researchers observed a dose-response relationship with smoking intensity with the highest risk of breast cancer in nurses smoking > 20 pack-years (HR: 1.32; 95% CI: 1.12–1.55) and > 15 cigarettes/day (HR: 1.31; 95% CI: 1.11–1.56). Moreover, it was observed that women who smoked > 10 pack-years before first childbirth had the highest breast cancer risk (HR: 1.58; 95% CI: 1.20–2.10) [12]. Malik A. et al. examined the association between passive smoking and breast cancer risk. Out of 100 breast cancer cases, 23% were exposed to secondhand smoke. Among passive smokers, 34.8% had exposure to tobacco smoke for ≥ 10 years during childhood, 65.2% were exposed for ≥ 20 years in adulthood. Authors suggested positive association between the intensity and duration of smoking and the age at presentation and poor prognostic factors [13]. In a Chinese case/control study Tong JH et al. found the relationship between passive smoking exposure from partners and breast cancer risk. Women whose partner smoked cigarettes had an increased risk of breast cancer (OR: 1.46; 95% CI: 1.05–2.03; P = 0.027). Moreover, it was observed that patients who smoked > 5 cigarettes/day also had significant increased risk (OR: 1.99; 95% CI: 1.28–3.10; P = 0.002), as were patients exposed to environmental tobacco smoke for 16–25 years (OR: 1.87 95% CI: 1.22–2.86; P = 0.004), and patients smoked > 4 pack-years (OR: 1.71 95% CI: 1.17–2.50; P = 0.004) [14]. In a large prospective cohort study the European Prospective Investigation into Cancer and Nutrition, it was observed that both active and passive smoking increase the risk of breast cancer, and smoking in the period between the first menstruation and the first pregnancy is particular-

ly dangerous. The findings indicated that 56.7% of the women surveyed never smoked, 23.3% were former smokers, and 20% of women smoked at the beginning of the study period. Of the surveyed woman 42.6% were exposed to second-hand smoke, 14.2% were not exposed to environmental tobacco smoke (neither at work nor at home), 7.1% were current or former smokers, never exposed to second-hand smoke, and 36.1% were active smokers exposed to second-hand smoke. In our study 67% of respondents were exposed to second-hand smoke. The risk ratio was similar for current (HR: 1.06; 95% CI: 1.00–1.12) and former (HR: 1.05; 95% CI: 1.00–1.10) smokers compared to women who had never smoked [15].

Conclusions

Exposure to environmental tobacco smoke increases the risk of breast cancer. Unfortunately, many women are exposed to secondhand smoke. This exposure also occurs at home, women are most often exposed to passive smoking by their husband/partner. Public awareness of the negative consequences of passive smoking should be raised. The undoubted strengths of the study are: (1) quite a large study group, (2) an extensive questionnaire and many analyzed variables, (3) wide range of smoking definitions. The study also has a number of weaknesses. The weaknesses of the study include the fact that: (1) all data are self-reported by the respondents, which is difficult to verify, (2) questionnaire questions concerned sometimes distant events in the past of the surveyed women, (3) relatively small numbers of respondents in the detailed definitions of smoking, which did not allow for a further division of these groups.

Funding: Data collection was carried out as part of research funds for the development of young researchers and PhD students at the Medical University of Lodz 502-03/6-024-0/502-64-100-18 and co-funded by Medical University of Lodz 503/6-024-01/503-66-001 and 503/6-024-01/503-61-001-19-00.

References

1. Eurostat Database. Smoking of tobacco products by sex, age, educational attainment level and degree of urbanization; 2020 [Internet]. Available from: https://appsso.eurostat.ec.europa.eu/nui/show.do?dataset=hlth_ehis_sk1e&lang=en [cited 01.08.2020].

2. Eurostat Database [Internet]. Quality report of the second wave of the European Health Interview survey, 2018 edition. Statistical report, Eurostat; 2018 [Internet]. Available from: https://ec.europa.eu/eurostat/documents/7870049/8920155/KS-FT-18-003-EN-N.pdf/eb85522d-bd6d-460d-b830-4b2b49ac9b03 [cited 01.08.2020].

3. Kaleta D, Korytkowski P, Makowiec-Dąbrowska T. Smoking in Poland in the population of professionally active people. Medycyna Pracy 2013; 64(3). https://doi.org/10.13075/mp.5893.2013.0031.

4. World Health Organization. Geneva; 2020 [Internet]. Available from: https://www.who.int/health-topics/tobacco [cited 13.01.2020].

5. West R. Tobacco smoking: Health impact, prevalence, correlates and interventions. Psychol Health 2017 Aug; 32(8): 1018–1036. https://doi: 10.1080/08870446.2017.1325890.

6. U.S. Department of Health, Washington; 1964. Education, and Welfare. Smoking and Health: Report of the Advisory Committee to the Surgeon General of the Public Health Service [Internet]. Available from: https://biotech.law.lsu.edu/cases/tobacco/nnbbmq.pdf [cited 13.01.2020].

7. Secretan B, Straif K, Baan R, Grosse Y, El Ghissassi F, Bouvard V et al. A review of human carcinogens – Part E: Tobacco, areca nut, alcohol, coal smoke, and salted fish. Lancet Oncol 2009; 10(11): 1033–1034. https://doi.org/10.1016/s1470-2045(09)70326-2.

8. Bray F, Ferlay J, Soerjomataram I, Siegel RL, Torre LA, Jemal A, Laversanne M. Global cancer statistics 2018: GLOBOCAN estimates of incidence and mortality worldwide for 36 cancers in 185 countries. CA Cancer J Clin 2018 Nov; 68(6): 394–424. https://doi: 10.3322/caac.21492.

9. Wojciechowska U, Didkowska J, Michałek I, Olasek P, Ciuba A. Cancer in Poland in 2018. Warsaw: Ministry of Health; 2020 [Internet]. Available from: http://onkologia.org.pl/wp-content/uploads/Nowotwory_2018.pdf [cited 19.05.2021].

10. Szkiela M, Kusideł E, Makowiec-Dąbrowka T, Kaleta D. Night Shift Work – A Risk Factor for Breast Cancer. Int J Environ Res Public Health 2020; 17(2): 659. https://doi.org/10.3390/ijerph17020659.

11. Macacu A, Autier P, Boniol M, Boyle P. Active and passive smoking and risk of breast cancer: a meta-analysis. Breast Cancer Res Treat 2015 Nov; 154(2): 213–224. https://doi: 10.1007/s10549-015-3628-4.

12. Andersen ZJ, Jørgensen JT, Grøn R, Brauner EV, Lynge E. Active smoking and risk of breast cancer in a Danish nurse cohort study. BMC Cancer 2017; 17: 556. https://doi.org/10.1186/s12885-017-3546-4.

13. Malik A, Jeyaraj PA, Shankar A, Rath GK, Mukhopadhyay S, Kamal VK. Passive Smoking and Breast Cancer – a Suspicious Link. Asian Pac J Cancer Prev 2015; 16(14): 5715–5719. https://doi: 10.7314/apjcp.2015.16.14.5715.

14. Tong JH, Li Z, Shi J, Li HM, Wang Y, Fu LY et al. Passive smoking exposure from partners as a risk factor for ER+/PR+ double positive breast cancer in never-smoking Chinese urban women: a hospital-based matched case control study. PLoS One 2014. https://doi.org/10.1371/journal.pone.0097498.

15. Dossus L, Boutron-Ruault MC, Kaaks R, Gram IT, Vilier A, Fervers B et al. Active and passive cigarette smoking and breast cancer risk: results from the EPIC cohort. Int J Cancer 2014; 134(8): 1871–1888. https://doi.org/10.1002/ijc.28508.

Journal of Health Study and Medicine
2021, No. 2
pp. 61-80
DOI 10.36145/JHSM2021.10

Perception of Advertising of Tobacco Products and Warnings about the Harmfulness of Smoking by Smokers

Ewa Siwińska-Beck[1]

Małgorzata Znyk[1]
https://orcid.org/0000-0002-0872-7293

Dorota Kaleta[1]
https://orcid.org/ 0000-0001-8453-8235

[1] Department of Hygiene and Health Promotion, Medical University of Lodz, Poland

Address for correspondence

Małgorzata Znyk
Department of Hygiene and Health Promotion, Medical University of Lodz
7/9 Zeligowskiego St., 90-752 Lodz, Poland
znyk@poczta.onet.pl

Abstract

Introduction: *Advertising of tobacco products contributes to the increase in the number of smokers. Also in Poland, the regulations in force prohibit advertising and promotion of tobacco products, symbols related to their use, as well as tobacco props and products imitating them. This prohibition applies to the promotion and advertising of tobacco products, including in public places, schools, health care institutions, but also on television, radio, in the press, cinemas and on billboards. The 6 effective actions of the MPOWER package of the World Health Organization, which may limit the consumption of tobacco products in society, include the elimination of marketing practices violating the ban on promotion and advertising of tobacco products, as well as warning and informing about health risks related to the use of tobacco. According to research, graphic health warnings on tobacco products are effective. They reliably reach tobacco users every time they use these products.*

Aim: *The aim of the study was to assess the perception of advertisements for tobacco products and warnings about the harmful effects of smoking by smokers.*

Material and methods: *A survey conducted in 2015. 114 smokers from Piotrków County. The research tool was a questionnaire. The Bioethics Committee of the Medical University of Lodz issued a positive opinion on the study (RNN/243/15/KE).*

Results: *During the last 30 days, smokers most often encountered information on the harmful effects of smoking on television (54%) and in newspapers or magazines (52%).*

Within the last 30 days, 97% of respondents saw information on the dangers of smoking on cigarette packets. 54% of smokers, under the influence of health warnings on cigarette packets, considered stopping smoking in the last 30 days. Smokers noticed forms of promotion of cigarettes: as many as 16% of respondents noticed free cigarette samples, 13% of discounted cigarettes. 36% of smokers believed that advertising and displaying tobacco products at points of sale did not affect the type of product purchased.

Conclusions: *There is a need for interventions to alert against the dangers of smoking. Rules to ban tobacco display and smoke warning can help reduce smoking.*

Key words: *tobacco advertising, media, tobacco outlets, smoking tobacco*

Introduction

The tobacco epidemic remains one of the main threats to public health, accounting for around 8 million deaths annually worldwide [1]. In Poland, tobacco consumption is also a public health concern [2]. Smoking in Poland is the most important risk factor responsible for the loss of 2 060 000 years of healthy life, which constitutes 16.3% of the total value of disability adjusted life-year (DALY) [3]. Every fifth Pole admitted to heavy smoking in 2019 [4].

The tobacco industry uses tobacco promotion, advertising and sponsorship (TAPS) as common tactics to encourage the use of tobacco products [5].

Low enforcement of the tobacco advertising ban, observed in many countries, makes it possible for the tobacco industries to promote tobacco products illegally.

Anti-smoking policy include a comprehensive ban on tobacco promotion and advertising and a smoking ban in many indoor public places. Anti-smoking policies were first introduced in the 1990s [2].

The 6 effective actions of the MPOWER package of the World Health Organization, which may limit the consumption of tobacco products in society, include the elimination of marketing practices violating the ban on promotion and advertising of tobacco products, as well as warning and informing about health risks related to the use of tobacco [6].

65% of the population (about 5 billion people) have at least one comprehensive smoking control measure. One of the methods introduced in around 91 countries is the conspicuous graphical health warnings on tobacco packages [1].

Also in Poland, the regulations in force prohibit advertising and promotion of tobacco products, symbols related to their use, as well as tobacco props and products imitating them. This prohibition applies to the promotion and advertising of tobacco products, including in public places, schools, health care institutions, but also on television, radio, in the press, cinemas and on billboards [7]. People's perception of the harmfulness of tobacco and nicotine products can influence their use.

Upon joining the Framework Convention on Tobacco Control (FCTC), Poland undertook to inform people who use tobacco products about the health effects of smoking and about the deadly threat posed by their use [8]. The Act of 1995 also imposes an obligation to inform about the harmfulness of tobacco smoking on the packaging of tobacco products [7].

The inclusion of health information and properly designed warnings on tobacco product packages reduces tobacco consumption.

Research shows that graphic health warnings on tobacco product packages are an effective form and reliably reach smokers every time they use them [8]. It is important that the warnings on the packages are large and cover at least half the surface of the tobacco package [9].

Health warnings and information containing both pictures and text are much more effective than purely textual [8].

In Poland, the unit packaging and collective packaging of smoking tobacco products bear the following warning: "Smoking kills – quit now!". Information is also provided where consumers can find out about support facilities for people who want to quit smoking (telephone numbers, website addresses, e-mail addresses) [7].

There is evidence that accurate, clear warnings make tobacco users think about quitting and may result in a reduction in tobacco use [10, 11].

The aim of the study was to assess the perception of advertisements for tobacco products and warnings about the harmful effects of smoking by smokers.

Materials and methods

The Bioethics Committee of the Medical University of Lodz approved the study (RNN/ 243/15/KE). In 2015 a cross-sectional study was carried out on smokers from the Piotrków district who gave their written consent to participate in the study. A detailed description of the study has been published elsewhere [12, 13].

The research tool was a questionnaire. The questionnaire consisted of the sections: information about the participant (socio-demographic data)

and information about tobacco (at points of sale, media, advertising). The tobacco information section provided data on how smokers had perceived advertising over the past 30 days. The perception of health warnings on cigarette packets that smokers have come into contact with in the last 30 days is also taken into account.

Results

114 study participants were cigarette smokers. Out of 114 respondents, 63 people (55%) were women, and 51 (45%) were men.

The most common smokers were married people (47%), in the 50–59 age group (33%), with secondary education (39%). 60% of the respondents worked as an employed person, and every fourth respondent had a net monthly income more than PLN 1,000 to PLN 1,500 per family member (Table 1).

Table 1. Characteristics of the studied population (N = 114)

Variable	N	%
Sex		
• female	63	55.3
• man	51	44.7
Age (years)		
• < 30	14	12.3
• 30–39	27	23.7
• 40–49	9	7.9
• 50–59	38	33.3
• ≥ 60	26	22.8
Marital status		
• bachelor/miss	28	24.6
• married	53	46.5
• divorced	18	15.8
• widower/widow	15	13.1
Education		
• basic	2	1.8
• basic vocational	21	18.4
• average	44	38.6
• post-secondary	14	12.3
• higher	33	28.9

Professional status in the last 12 months		
• salaried employee	68	59.7
• self-employed person	11	9.6
• pupil/student	3	2.6
• housewife	2	1.8
• pensioner / annuitant	23	20.2
• unemployed	7	6.1
Monthly net family income per person		
• up to 500 PLN	11	9.7
• over 500 to 700 PLN	8	7.0
• over 700 to 1000 PLN	18	15.8
• above 1000 to 1500 PLN	29	25.4
• above 1500 to 2000 PLN	26	22.8
• above 2000 to 2500 PLN	13	11.4
• above 2500 PLN	9	7.9
Smoking tobacco		
• Yes everyday	106	92.9
• Yes, less than every day	8	7.1
How many cigarettes do you smoke in total during the day? (pieces)		
• < 1	2	1.7
• 1–5	5	4.4
• above 5 do 10	27	23.7
• above 10 do 20	61	53.5
• above 20 do 30	18	15.8
• above 30	1	0.9
Number of years of regular daily smoking? (after deducting any interruptions for abstinence)		
• < 10	17	15.0
• 10–20	36	31.6
• 21–30	29	25.4
• 31–40	20	17.5
• > 40	11	9.6
• No data	1	0.9
Have you ever tried to quit smoking?		
• Yes	65	57.0
• No	49	43.0
If you have ever tried to quit smoking it how many times?		
• 0	2	1.7
• 1–2	37	32.5
• 3–4	30	26.3
• 5–6	6	5.3
• 6+	3	2.6
• No data	36	31.6

7% are smokers less frequently than daily smokers, and 93% of all respondents are daily smokers. The fewest (0.9%) smoked more than 30 cigarettes during the day, and the most common (53%) more than 10 to 20 cigarettes.

The number of years of regular daily smoking for every third subject was 10–20 years, and for every fourth subject – 21–30 years (after subtracting any interruptions for abstinence).

65 respondents (57%) have ever tried to quit smoking, the most common being 1–2 attempts in their life (33%).

Television, newspapers and magazines are the places where smokers most often encountered information on the harmfulness of smoking in the last 30 days (54% and 52% respectively). Educational and health materials (45%), the Internet (38%), billboards (25%) and radio (24%) were also often mentioned as sources of information on the harmfulness of smoking.

During the last 30 days, 110 respondents (97%) saw information on the dangers of smoking on cigarette packets. 62 smokers (54%) considered quitting smoking in the past 30 days because of perceived health warnings on cigarette packets.

Most of the respondents (47%) were in favor of introducing graphic forms on tobacco products showing the harmful effects of smoking, while 22% were against it.

Smokers noticed various forms of cigarette promotion: 16% of respondents noticed free cigarette samples, 13% of discounted cigarettes, 8% of clothes or other articles with the name or logo of the cigarette brand. 8% of smokers noticed offers of free gifts and discounts on other products when buying cigarettes. 7% of respondents saw promotions of cigarettes in postal items (Table 2).

Table 2. Smokers' perception of tobacco advertising (N = 114)

Variable	N	%
Are in the last 30 days in the following media you have come across the information about the dangers of smoking or those that have encouraged you to quit smoking?		
In newspapers or magazines		
• Yes	59	51.8
• No	50	43.8
• Not applicable	5	4.4
In TV		
• Yes	61	53.5
• No	50	43.9
• Not applicable	3	2.6
On the radio		
• Yes	27	23.7
• No	83	72.8
• Not applicable	4	3.5
On billboards		
• Yes	28	24.6
• No	83	72.8
• Not applicable	3	2.6
Online		
• Yes	43	37.7
• No	66	57.9
• Not applicable	5	4.4
In educational and health materials		
• Yes	51	44.7
• No	59	51.8
• Not applicable	4	3.5
In the last 30 days, have you seen information on the dangers of smoking on cigarette packets?		
Yes	110	96.5
No	4	3.5
In the last 30 days, have you considered stopping smoking due to the health warnings on cigarette packets?		
Yes	62	54.4
No	52	45.6

In the last 30 days, have you noticed any of the following forms of cigarette promotion?		
Free cigarette samples		
• Yes	18	15.8
• No	85	74.6
• I don't know	11	9.6
Cigarettes on sale		
• Yes	15	13.2
• No	88	77.2
• I don't know	11	9.6
Cigarette tickets		
• Yes	5	4.4
• No	97	85.1
• I don't know	12	10.5
Offers of free gifts or discounts on other products when buying cigarettes		
• Yes	9	7.9
• No	91	79.8
• I don't know	14	12.3
Clothes or other items with a name or logo cigarette brands		
• Yes	9	7.9
• No	93	81.6
• I don't know	12	10.5
Promotion of cigarettes in postal items		
• Yes	8	7.0
• No	93	81.6
• I don't know	13	11.4
Do you think that advertising and displaying tobacco products at points of sale make it difficult for you to quit smoking?		
• Definitely not	25	21.9
• Probably not	48	42.1
• I have no opinion	23	20.2
• Probably yes	12	10.5
• Definitely yes	6	5.3

Do you think that advertising and displaying tobacco products at points of sale affect the choice of the brand of the product you buy?		
Definitely not	24	21.0
Probably not	46	40.4
I have no opinion	18	15.8
Probably yes	20	17.5
Definitely yes	6	5.3
Do you think that advertising and displaying tobacco products at points of sale affect the type of product you buy?		
Definitely not	20	17.5
Probably not	41	36.0
I have no opinion	22	19.3
Probably yes	25	21.9
Definitely yes	6	5.3
Do you consider it right to raise the tax on tobacco products?		
Definitely not	33	29.0
Probably not	43	37.7
I have no opinion	28	24.6
Probably yes	8	7.0
Definitely yes	2	1.7
Do you consider it right to prohibit the display of tobacco products in points of sale?		
Definitely not	12	10.5
Probably not	41	36.0
I have no opinion	36	31.6
Probably yes	17	14.9
Definitely yes	8	7.0
Do you consider it right to introduce a complete ban on the production and sale of cigarettes and other tobacco products intended for smoking?		
Definitely not	45	39.5
Probably not	44	38.6
I have no opinion	19	16.7
Probably yes	3	2.6
Definitely yes	3	2.6

Do you consider it right to introduce pictures of the harmful effects of smoking on tobacco products?		
Definitely not	15	13.2
Probably not	17	14.9
I have no opinion	28	24.6
Probably yes	29	25.4
Definitely yes	25	21.9

According to the surveyed smokers, 36% believed that advertising and displaying tobacco products at points of sale did not affect the type of product they buy, while 18% believed that it definitely did not. Every fifth person did not have an opinion on this subject.

For 22% of point-of-sale smokers, it would be appropriate to ban the display of tobacco products. Every third person did not have an opinion on this subject, and 46% did not share this opinion.

For the majority of respondents (about 64%), displaying tobacco products and advertising at points of sale did not make it difficult for smokers to quit smoking.

5% of respondents were in favor of introducing a complete ban on the production and sale of cigarettes and other tobacco products, 79% of respondents did not support this ban. 67% of respondents were in favor of raising the tax on tobacco products.

Discussion

Our research complements the literature on the perception of tobacco advertising by smokers and warnings about the dangers of smoking.

Points of sale (POS) have become important tobacco display venues for tobacco companies in countries where tobacco advertising and promotion bans have been introduced [14].

Our study found that smokers notice various forms of cigarette promotion. Free cigarette samples (16%) and discounted cigarettes (13%) were most often perceived. Offers of free gifts and discounts on other products were also noted at points of sale when buying cigarettes (8%).

This may indicate poor enforcement of the ban on tobacco advertising at points of sale. Moreover, they show that enforcement of existing legislation needs to be improved.

Other studies also show a strong correlation between smoking initiation, susceptibility and progression and exposure to advertising (including at POS) [14–20].

Every second person in our study did not support the validity of the ban on displaying tobacco products at POS (points of sale). It was observed that when shopping at POS, noticing tobacco at an exhibition each time more than threefold increased the likelihood of susceptibility compared to never noticing tobacco [16].

Every fourth smoker, as a result of viewing a cigarette exhibition, bought cigarettes at least sometimes on an impulse. One in eight smokers who have recently quit smoking and one in five smokers who have tried to quit, avoided outlets where they usually bought cigarettes. These were actions in case they were tempted to buy them [18].

34% of people who have recently quit smoking and 38% of smokers who have tried to quit in the last 12 months have experienced an urge to buy cigarettes after viewing a retail display [18]. Another research by Kaleta et al. carried out in Poland showed a violation of the ban on tobacco and e-cigarette advertising in POS. There was a decrease in the promotion of tobacco products at POS in the form of boards, mats and posters, but an increase in video screens (8% in 2014, 30% in 2019). Accessories and products imitating tobacco products were less popular TAPS (10% in 2014, 2% in 2019) [21].

In another study, which was realized in 25 European countries, exposure to indirect advertising such as free cigarette samples or tobacco company logos on promotional items was common in all countries.

10.9% said that a tobacco company representative had ever offered them "free" cigarettes. The item with the cigarette brand logo was owned by 18.2% of respondents. 20% of respondents in Poland, the Republic of Serbia and Montenegro were offered free cigarettes [22].

In contrast, a study by Rudatsikira et al found that at least 95.5% of men and women reported seeing a tobacco brand on TV in the last

30 days. About half of the study participants reported tobacco-related advertisements in magazines in the past 30 days, and 7.8% reported possession of an item with a tobacco brand logo. Women saw tobacco-related advertisements in the media more often than men [23].

Free tobacco products and tobacco advertising on television, on video, in shops and at street vendors are more likely to use tobacco [24]. A study in 19 developing countries showed that there is a positive correlation between smoking and exposure to advertising [25].

Smokers in the display ban conditions were less likely to attempt to purchase cigarettes. People who recently quit smoking under the advertising ban were less likely to smoke [26]. Many smokers (31.4%) believe that removing cigarette displays from shops will make it easier for them to quit [18].

The main goal of tobacco control policy is to communicate the dangerous effects of smoking. In our study, 45% of respondents came across information on the harmfulness of tobacco smoking in educational and health materials, and 38% on the Internet. More than half of the smokers in our study most often encountered information on the harmful effects of smoking on television, as well as in newspapers or magazines.

It has been shown that, in addition to reducing the advertising of tobacco products, warning signs and less attractive cigarette packages have been shown to reduce tobacco smoking [27]. Our study showed that as many as 97% of smokers saw information on cigarette packets warning against the harmful effects of smoking. Similar results were obtained in other studies (over 90% of respondents) [28–30].

Research shows that smoking cessation or smoking reduction can be influenced by warning labels placed on tobacco products [28, 31].

This proves the effectiveness of displaying graphic health warnings on the packages of tobacco products [1, 32].

In another study, 95% of respondents were informed about the harmful effects of smoking. Under the influence of this information, 14% of people reduced the number of cigarettes smoked per day, and 5% intended to quit smoking. The majority (47%) believed that health warnings with images and text could have a greater impact than text-only warnings [33].

Another study confirms that the warnings perceived by respondents (70%) increase the awareness of the health risks associated with smoking. They also help to reduce or quit smoking [28].

Respondents notice warnings on tobacco products, and most believe that they can understand warning labels [30]. Smokers exposed to plain packets try to buy cigarettes much less frequently than people exposed to colored packets [26].

In our study, every second smoker believed it was right to introduce pictures of the harmful effects of smoking on tobacco products. In other studies, all picture warnings on cigarette packets were found to be more effective than text warnings [30, 34]. Pictorial health warnings are seen to be more effective in stopping smoking among non-smokers as well as in stimulating smoking cessation among smokers [30, 35]. They are eye-catching and provide additional motivation to quit smoking [30].

Every second smoker in our study, under the influence of health warnings on cigarette packets, considered giving up smoking in the last 30 days.

Also in the study by Sharm et al., a significant relationship was found between awareness of the negative effects of smoking and an attempt to quit smoking [36].

Our study is a study that was first conducted among the socially disadvantaged rural adult population. The analysis has strengths and weaknesses that have been described elsewhere [12]. The study used a cross-sectional design that tends to be observable at one point in time, making it impossible to observe changes over longer periods of time. The study is also limited by a small group of the population.

Conclusions

There is a need for interventions to alert against the dangers of smoking. Rules to ban tobacco display and smoke warning can help reduce smoking. A policy that introduces warning labels on the packaging of tobacco products can effectively inform people about the adverse health effects of smoking and play a key role in reducing the number of people smoking.

References

1. World Health Organization. WHO Report on the Global Tobacco Epidemic 2019: Offer Help to Quit Tobacco Use [Internet]. Available from: https://www.who.int/teams/health-promotion/tobacco-control/who-report-on-the-global-tobacco-epidemic-2019 [cited 10.05.2021].

2. OECD/European Observatory on Health Systems and Policies. Country Health Profile, Poland; 2019; State of Health in the EU, OECD Publishing, Paris/European Observatory on Health Systems and Policies, Brussels, Belgium; 2019 [Internet]. Available from: https://doi.org/10.1787/297e4b92-en [cited 10.05.2021].

3. Wojtyniak B, Gorynski P. Health Status of Polish Population and Its Determinants. Warsaw: National Institute of Public Health National Institute of Hygiene; 2020.

4. Raport Głównego Inspektoratu Sanitarnego. Postawy Polaków wobec palenia tytoniu. GIS; 2019 [Internet]. https://www.gov.pl/web/gis/postawy-polakow-wobec-palenia-tytoniu--raport-2017.

5. Gautam P, Bhatta D, Sharma E, Rahman A, Dawit R, Li W, Ebrahimi Kalan M, Acharya Gautam S, Li T, Maziak W. Influence of Tobacco Marketing on Nepalese Adolescents: Cigarette Use and Susceptibility to Cigarette Use. Asian Pac J Cancer Prev 2020; 21(9): 2689–2695. https://doi.org/10.31557/APJCP.2020.21.9.2689.

6. Program ograniczenia zdrowotnych następstw palenia tytoniu w Polsce. Cele i zadania na lata 2014–2018. Warszawa: Ministerstwo Zdrowia; 2013.

7. Act of 9 November 1995 on the Protection of Health from the Consequences of Tobacco (Dz.U. 2021, poz. 276) [Internet]. Available from: https://isap.sejm.gov.pl/ [cited 10.05.2021].

8. Ramowa Konwencja Światowej Organizacji Zdrowia (WHO) o ograniczeniu palenia tytoniu. Genewa: WHO Regional Office for Europe; 2009.

9. Kowitt SD, Noar SM, Ranney LM, Goldstein AO. Public attitudes toward larger cigarette pack warnings: Results from a nationally representative U.S. sample. PLoS One 2017; 12(3): e0171496.

10. Kamyab K, Nonnemaker JM, Farrelly MC. Public support for graphic health warning labels in the U.S. American Journal of Preventive Medicine 2015; 48(1): 89–92.

11. Policy Evaluation. FCTC Article 11: tobacco warning labels. Evidence and recommendations from the ITC project. Waterloo, Ontario: International Tobacco Control; 2009.

12. Znyk M, Kaleta D. Subjective assessment of health in people with varying degrees of nicotine addiction. Journal of Health Study and Medicine 2020; 4: 47–65. https://doi.org/10.36145/JHSM2020.26.

13. Milcarz K, Makowiec-Dąbrowska T, Bąk-Romaniszyn L, Kaleta D. Smoking Patterns and Smoking Cessation Willingness-A Study among Beneficiaries of Government Welfare Assistance in Poland. Int J Environ Res Public Health 2017; 14: 131.

14. Ford A, MacKintosh AM, Moodie C, Kuipers MAG, Hastings GB, Bauld L. Impact of a ban on the open display of tobacco products in retail outlets on never smoking youth in the UK: findings from a repeat cross-sectional survey before, during and after implementation. Tob Control 2020; 29(3): 282–288.

15. Usidame B, Miller EA; Cohen JE. Assessing the Relationship between Retail Store Tobacco Advertising and Local Tobacco Control Policies: A Massachusetts Case Study. J Environ Public Health 2019; 1823636.

16. Spanopoulos D, Britton J, McNeill A, Ratschen E, Szatkowski L. Tobacco display and brand communication at the point of sale: Implications for adolescent smoking behaviour. Tob Control 2014; 23: 64–69.

17. Paynter J, Edwards R. The impact of tobacco promotion at the point of sale: A systematic review. Nicotine Tob Res 2009; 11: 25–35.

18. Wakefield M, Germain D, Henriksen L. The effect of retail cigarette pack displays on impulse purchase. Addiction 2008; 103: 322–328.

19. Petticrew M, Semple S, Hilton S, Creely KS, Eadie D, Ritchie D, Ferrell C, Christopher Y, Hurley F. Covert observation in practice: Lessons from the evaluation of the prohibition of smoking in public places in Scotland. BMC Public Health 2007; 7: 204.

20. World Health Organization. WHO Report on the Global Tobacco Epidemic, 2013. Enforcing Bans on Tobacco Advertising, Promotion and Sponsorship [Internet]. Available from: https://www.who.int/tobacco/global_report/2013/en/ [cited 10.05.2021].

21. Polanska K, Kaleta D. Tobacco and E-Cigarettes Point of Sale Advertising – Assessing Compliance with Tobacco Advertising, Promotion and Sponsorship Bans in Poland. Int J Environ Res Public Health 2021; 18(4): 1976. https://doi.org/10.3390/ ijerph18041976.

22. Baska T, Warren ChW, Baskova M, Jones NR. Prevalence of youth cigarette smoking and selected social factors in 25 European countries: findings from the Global Youth Tobacco Survey. Int J Public Health 2009; 54(6): 439–445. https://doi.org/10.1007/s00038-009-0051-9.

23. Rudatsikira E, Muula AS, Siziya S. Prevalence, Correlates of and Perceptions Toward Cigarette Smoking Among Adolescents in South Korea. Indian J Pediatr 2009; 76(5): 505–510.

24. Khan JR, Raheem E, Daniel M. The prevalence and correlates of tobacco use among 7–9th grade students in Bangladesh. Children and Youth Service Review 2019; 104: 104406.

25. Kostova D, Blecher E. Does advertising matter? Estimating the impact of cigarette advertising the impact of cigarette advertising on smoking among youth in developing countries. Contemporary Economic Policy 2013; 31(3). https://doi.org/10.1111/j.1465-7287.2012.00323.x.

26. Nonnemaker J, Kim A, Shafer P, Loomis B, Hill E, Holloway J, Farrelly M. Influence of point-of-sale tobacco displays and plain black and white cigarette packaging and advertisements on adults: Evidence from a virtual store experimental study. Addictive Behaviors 2016; 56: 15–22.

27. Asamura H, Chansky K, Crowley J, Goldstraw P, Rusch VW, Vansteenkiste JF. The International Association for the Study of Lung Cancer Staging Project: proposals for the revision of the N descriptors in the forthcoming 8th edition of the TNM classification for lung cancer. J Thorac Oncol 2015; 10(12): 1675–1684. https://doi.org/10.1097/JTO.0000000000000678.

28. Chopra A, Rao NC, Gupta N, Vashisth S. Communicating tobacco health risks: How effective are the warning labels on tobacco products? Niger Med J 2014; 55(5): 411–416. https://doi.org/10.4103/0300-1652.140383.

29. Shah VR, Dave VR, Sonaliya KN. Impact of anti-tobacco warning labels on behaviour of tobacco users in one of the cities of Gujarat, India. J Prev Med Hyg 2013; 54(2): 109–113.

30. Vanishree N, Narayan RR, Naveen N, Bullapa D, Vignesh D, Raveendran NMP. Impact of pictorial warning labels on tobacco products among patients attending outpatient department of a dental college in Ban-

galore city: A cross-sectional study. Indian J Cancer 2017; 54(2): 461––466. https://doi.org/10.4103 / ijc.IJC_203_17.

31. Chow CK, Corsi DJ, Gilmore AB, Kruger A, Igumbor E, Chifamba J et al. Tobacco control environment: cross-sectional survey of policy implementation, social unacceptability, knowledge of tobacco health harms and relationship to quit ratio in 17 low-income, middle-income and high-income countries. BMJ Open 2017; 7(3): e013817. https://doi.org/10.1136/bmjopen-2016-013817.

32. Borland R, Wilson N, Fong GT, Hammond D, Cummings KM, Yong HH, Hosking W, Hastings G, Thrasher J, McNeill A. Impact of graphic and text warnings on cigarette packs: findings from four countries over five years. Tob Control 2009; 18(5): 358–364.

33. Mannocci A, Antici D, Boccia A, Torre GL. Impact of cigarette packages warning labels in relation to tobacco-smoking dependence and motivation to quit. Epidemiol Prev 2012; 36(2): 100–107.

34. Alaouie H, Afifi RA, Haddad P, Mahfoud Z, Nakkash R. Effectiveness of pictorial health warnings on cigarette packs among Lebanese school and university students. Tob Control 2015; 24(e1): e72–80. https://doi.org/10.1136/tobaccocontrol-2012-050745.

35. Suci PR, Dewi S. Perceived effectiveness of pictorial health warnings on changes in smoking behaviour in Asia: a literature review. BMC Public Health 2018; 18(1): 1165. https://doi.org/10.1186/s12889-018-6072-7.

36. Sharma SK, Prasuna G. Thakur K, Gupta M. Effect of magnification and changes in tobacco pictorial warning on asceticism of tobacco use: An exploratory survey. J Family Med Prim Care 2020; 9(12): 6051–6060. https://doi.org/10.4103/jfmpc.jfmpc_1 509_20.

Journal of Health Study and Medicine
2021, No. 2
pp. 81-103
DOI 10.36145/JHSM2021.11

Identity and Culture of Medical Schools in Poland

Joanna Sułkowska[1]
https://orcid.org/0000-0002-4949-3622

[1] Institute of Health Sciences, University of Social Sciences, Lodz, Poland

"Historiam nescire, hoc est semper purerum esse"
Cicero [1]

Address for correspondence

Joanna Sułkowska
Institute of Health Sciences, University of Social Sciences
121 Gdanska St., 90-519 Lodz, Poland
jsulkowska15@wp.pl

Abstract

A scientific school could be defined as a form of community that includes a group of scholars, researchers sharing similar ideas, and approaching specific scientific problems in the same way. The crucial characteristic of a scientific school is based on values, culture and identity of scholars represented by similar research orientation.

The objective of the paper is to describe the characteristics of the organizational identity of medical science schools. The research methodology is based on a review of the literature on the subject.

Key words: *organizational identity, organizational culture, medical schools*

Introduction

Organizational identity is an interdisciplinary issue undertaken by scientists working in many disciplines: management sciences, economics, organizational theory, and sociology. Scientific schools are examples of organizations that are characterized by a strong identification with the issues they undertake, a similar methodology, and the scientific authorities creating them. The participants of the science school identify with similar values, adhere to similar norms and create strong organizational cultures [74, 75, 76]. They have common ideas and carry out joint ventures. It is particularly noticeable in medical disciplines where, from the beginning of the educational and professional path, the master-student relationship is of great value and the basis of education.

It can be argued that scientific schools related to research, publication achievements and scientific authorities are established in various research centers. The aim of the work is to describe the characteristics of the organizational identity of medical science schools. The research methodology is based on a review of the literature on the subject.

Definitions of scientific schools

An attempt to define the concept of a scientific school is an interdisciplinary meeting of representatives of various fields of science: history, philosophy, sociology, and linguistics. It can be assumed that each field of knowledge needs to look back at the achievements of its predecessors. A scientific school is defined as a form of community that includes a group of scholars, researchers sharing similar ideas, and approaching specific scientific problems in the same way. Schools are associated with a given place or research center, and are often an attempt to continue the achievements of an outstanding scientist. Researchers who become elements of a given research unit also have a similar scientific approach. In the literature on the subject, you can find cases of scientific schools that are named and defined in terms of time, and their achievements

constitute a form of a collection of research activities of scientists included in them. They are scientists with recognized, often international achievements, authors of groundbreaking works, creators of epochal inventions, or originators of new theories. The followers are the continuators and successors of the thoughts of their predecessors. There is a characteristic sense of internal solidarity, as well as the need to defend against attacks by scientific opponents from other centers. The establishment of each science school has its own historical background. These are organizations that arise and operate in specific circumstances. Schools are subject to many transformations, they evolve, they collapse, they change. They are replaced by other individuals who draw on their legacy, broadening the spectrum of their achievements or even negating their achievements. Newly created entities may become modifications of previously existing schools [2]. In the figures of the masters and their achievements, one can see attempts to define the community they create. In the history of science, there are many examples of opinion-forming authorities who became the progenitors of scientific schools later named after them. Epicurus (341 BCE–270 BCE) one of the most important Greek philosophers who, after arriving in Athens, founded around 306 BCE the „Garden" philosophical school, thus giving rise to an elite community of scientists, to which belonged Amynomachos from Athens, Metrodoros from Lampsacus (approx. 330–277 BC), Polyainus from Lampsacus (approx. 340 BC – died approx. 278 BC), Hermarchos of Mytilene, Philodemus of Gadara (approx. 110 BC – approx. 39 BC), Zeno of Sidon (approx. 150 BC – approx. 70 BC) [3]. It gave rise to a philosophical trend that was continued in Roman times by Titus Lucretius Carus (c. 99 BCE–55 BCE) and in the modern era, e.g. by the French philosopher – Pierre Gassendi (1592–1655) [4].

Among the definition of a scientific school, it is worth mentioning the one formulated by the sociologist Zbysław Muszyński, who describes it as an informal organization gathering a community of scientists that arises spontaneously and apart from the need to solve research problems, is also a sociological and psychological phenomenon of great importance

for the development of science. Muszyński considers the following criteria for describing such a community of scientists: "genealogy, time, place, self-awareness, community of views, writings, styles and the world view of researchers" [5]. The origin of the science school is the master-student relationship. A master is a person with significant scientific authority, outstanding personality and organizational skills, distinguished by the ability to cooperate and creatively influence others. It is a phenomenon located in time: it arises and functions at a specific moment. These two categories describing a science school allow the stages of its development to be traced: the constitution of the master; selection of students; the establishment of a school in terms of common ideas and methodology, development and scientific achievements, and the final stage of spreading the idea or terminating its activities.

The place is often traditionally associated with the school's seat as a research center. The members of a given community share the awareness of unity and a sense of separateness.

The concept of school may be identified in relation to the research issues it undertakes, e.g. a sociological school. By reviewing research centers, one can find their connections with the direction of research, e.g. a school of interpersonal relations, but also with their location, a university or a city where researchers gathered, giving them the opportunity to develop and act, e.g. the Harvard School, the Michigan School. Different scientific directions and approaches can be found in one research unit. Each of them has a specific epistemology and methodology of scientific conduct [6]. For the contemporary definition of scientific schools, the concept of paradigm is binding. It was formulated in the 18th century in Göttingen by Georg Christoph Lichtenberg (1742–1799), a professor of natural sciences. The term "paradigm" is derived from the Greek language parádeigma is a pattern, a model with a didactic value, allowing direct insight into the complexity of scientific research [7]. The foundations of the paradigm concept should be sought in the ancient Platonic idea, in which it was described as a model of variable things, from the Greek word [8].

In an attempt to search for the genesis of the definition of a scientific school, reference should also be made to Thomas Samuel Kuhn (1922––1996), the creator of the paradigm theory, who in his book: The Structure of Scientific Revolutions of 1962, presented the course of the revolution in science. Kuhn called the paradigm necessary to solve a specific research problem, which is accepted by a given scientific community [9]. It becomes a criterion identifying a specific field of science [10]. Kuhn believed that every scientific community at some point adopts a certain paradigm, taking as fundamental the foundations of a given scientific discipline [72, 73]. Therefore, it plays the role of a criterion describing a given field, enables an individual researcher to work within a given group of scientists and allows to define the framework of a scientific field [11].

History of medical science schools

The origins of the tradition of establishing scientific schools can be traced back to antiquity. In 387 B.C.E. Plato founded the Academy in Athens, where philosophy, mathematics, rhetoric and natural sciences were studied. The most eminent mathematicians of that time were active in it [12, 13]. Aristotle is considered to be the greatest continuator of the philosophical formation of Plato, the foundations of his philosophical reflections lie in the reflections of his teacher [14, 15]. The Platonic Academy had a great influence not only on the philosophy of this period, but also on the whole of ancient thinking. Its closure in 529 CE. by the Roman emperor Justinian, it is considered the moment separating the ancient epoch from the middle ages [16]. The successor of Plato's Academy was the Lyceum (Greek: Λύκειον) founded by Aristotle in the 4th century BC on the basis of existing from the 5th century BC at the temple of Apollo Likejos, gymnasium. It conducted research in many areas of knowledge, mainly in the humanities and natural sciences. The most eminent representatives of the school include: the father of botany, Theophrastus of Eresos (ca. 370–287 BC [17] and Aristoxenos of Taranto (ca. 354 BC–300 BC) [18]. Sotera, ruler of Egypt in 323–283 BC

[19], Mouseion, (Greek: Μουσεῖον) – a temple of muses, a scientific school of great importance to the ancient world, both in the humanities and the sciences [20]. The scientific schools include Demetrius of Phaleron (c. 350–283 BCE) [21] or Euclid (c. 365 BCE–270 BCE) [22].

In the Renaissance in Italy, schools began to appear, which later gave rise to scientific societies. One of the most outstanding is the Accademia Platonica in Florence, founded in 1462 under the patronage of Cosmas Medici in the Careggi villa [23]. The first scientific society, Academia Secretorum Naturae, was founded by Giambattista della Porta in Naples in 1560 [24]. In 1582, the Accademia della Crusca was founded in Florence, inspired by Anton Francesco Grazzini. The purpose of its creation was to keep the Italian language clean and in good condition [25]. In 1542, the Accademia Vitruviana was established in Rome by the Italian philologist Claudio Tolomei, and on August 17, 1603, the National Academy of Lynx in Rome. It was the first true modern scientific society. The main task of the school was to promote experimental natural science, and one of its outstanding members was an astronomer, mathematician, physicist and philosopher – Galileo Galilei (1564–1642), who supported the theory of Copernicus [26]. Also in Florence, the Accademia del Cimento – the Academy of Trials, dealing with physical sciences, was founded in 1657. Its model was used to create the Royal Society in London and the Academy of Sciences in Paris. Two years later, in 1677, the Accademia Fisico-Matematica [27] was established in Rome. Following the traditions of Italian science schools, similar institutions began to appear in other European countries, including Poland. The first was the Sodalitas Litteraria Vistulana-Nadwiślańskie Towarzystwo Literackie [28, 29], established in Cracow in 1489. The first Polish scientific school was the "Society of Writers in Poland for Publishing the Best and Most Beneficial Books of the Country" (Societas litteraria ad edendos optimos et Poloniae utilissimos libros, operating in 1765–1770 in Warsaw). On February 10, 1775, the Society for Books was also established in Warsaw. Its mission was to develop school curricula and publish school textbooks [31]. In 1800, the Society of Friends of Sciences (TPN) was established in Warsaw, which brought

together researchers from various fields of science. Its work contributed to the establishment of the Royal University of Warsaw, the precursor of the University of Warsaw [32]. On the other hand, in 1872, the Academy of Skills was established in Cracow, which was called the Polish Academy of Learning from 1919. Initially, it consisted of 3 faculties: philology, history and philosophy, and mathematics and natural sciences, and from 1930 also medicine [33].

The Lviv-Warsaw School can be considered the most famous Polish pre-war philosophical school. It was established by Kazimierz Jerzy Adolf Twardowski (1866–1937), the founder of the Polish Philosophical Society [34]. The school was established at the end of the 19th century in Lviv and was continued by his students in Warsaw in later years. The outbreak of World War II marked the end of its existence. However, the influence of the school and the methodology connected with it remained in the works of its continuators, continuing uninterruptedly until the 1950s [35].

The beginnings of the Polish school of philosophy of medicine are related to the publication in 1874 of the work of Tytus Chałubiński (1820––1889) "On the method of finding medical indications". The main field of interest of the researchers was the logic of diagnosis and the approach to the topic of prognosis in medicine. The most prominent representatives of the older Polish school of medical philosophy were: Tytus Chałubiński [36], Feliks Franciszek Nawrocki (1838–1902), Ignacy Baranowski (1833–1919), Henryk Nusbaum (1849–1937), Henryk Fryderyk Hoyer (1834–1907), Wiktor Feliks Szokalski (1811–1891) [38].

In 1805, the Vilnius Medical Society was established, the first president of which was the outstanding Polish scientist Jędrzej Śniadecki [39]. Its founders were the doctor August Becu (1771–1824), chemist Jędrzej Śniadecki (1766–1838), Józef Frank, Jan Braun, Eliasz Enholm, Herz Heiman, Jakób Liboszyc, Jana Lobenwein, Andrzej Matusewicz, Jana Szlegel, Ferdynand Spitznael and Jan Szymkiewicz. After the Vilnius University was closed in 1831, it existed thanks to cooperation with the Medical-Surgical Academy [40].

In December 1820, the Warsaw Medical Society was established. The Society was founded on the initiative of professors of the University of Warsaw: Andrzej Franciszek Ksawery Dybek (1783–1826), August Ferdynand Wolff (1768–1846), Józef Czekierski (1777–1827), Franciszek Brandt, Maurycy Woyd (1791–1877), Fryderyk Roemer, Jan Kuehne, Jan Theiner. In 1889, the Laryngological Section was established at the Warsaw Medical Society, constituting the foundations of the first scientific association of Polish otolaryngologists. Its chairman was Dr. Teodor Heryng (1847–1925) [41]. In 1908, the Laryngological Section was created at the Lviv Medical Society, and in 1909 it was transformed into the Lviv Otolaryngological Society, headed by prof. Antoni Jurasz senior. In 1912, the Warsaw Otolaryngological Society was established [42]. Ludwik Guranowski (1853–1926) and Zygmunt Silver (1860–1941) became its chairmen [43]. The following actors were active in it: Zdzisław Dmochowski, Teodor Heiman, Alfred M. Sokołowski, Leopold Lubliner, Jan Moczulski, Robert Sinołęcki [45]. In November 1924, on the initiative of Dr. Jan Pieniążek, the Łódź Section of the Otolaryngological Society was established [46]. The fire of war interrupted the work of the Society, but the tradition of meetings and exchange of experiences between specialists survived. The Łódź Branch of the Polish Otolaryngological Society was reactivated, on the initiative of the team members, in 1945, four months after the establishment of the Chair and Clinic of Ear, Nose, Throat and Larynx Diseases [47]. In 1961, at the request of associate professor Jan Danielewicz, the Pediatric Laryngology Section of PTORL was established, which is currently chaired by prof. Jarosław Szydłowski, and in 1963 in Wrocław the Audiological Section of PTORL was established, headed by prof. Wiesław J. Sułkowski. The structures of PTORL also include: Phoniatric Section chaired by prof. Jurek Olszewski, Historical Section working under the supervision of prof. Andrzej Kierzek, Oncology Section led by prof. Paweł Burduk, Section of Otology and Otoneurology operating under the direction of prof. Wioletta Pietruszewska, Section of Rhinology and Plastic Surgery of the Face – prof. Eliza Brożek-Mądry [48].

The identity and organizational culture of a medical scientific school

According to an encyclopedic definition, identity is a multi-meaning term referring to individuals or social groups which, despite being subject to numerous, inevitable changes, retain certain constant features that determine their identification, who or what they are. It is a feature that distinguishes an individual or a group from others, giving it its difference, describing it [49]. The word comes from the Latin word idem, which means identity, continuity. An attempt to define the concept has already been read among ancient philosophers. Aristotle in "Metaphysics" wrote that: "...identity is a certain unity of a multiplicity of things or a unity of one thing understood as a multiplicity...". Considerations on identity were then undertaken by: Leibniz, Kant, Hegel, and Heidegger [50]. In psychology, it is described in the context of perceiving oneself as separate and unique in relation to others. Since the emergence of the theory of the American psychoanalyst Erik Homburger Erikson (1901–1994), it has been defined in terms of two important human relationships: his relationship to himself and other people, and thus also to tradition and culture [51]. Social identity, on the other hand, is the identity of an individual or a community, thanks to which it locates itself in a specific area of social reality [52]. The contemporary concept of identity in relation to management science was developed by its creators: George Herbert Mead (1863–1931) and Herbert Blumer (1900–1987) [53]. In the review of the literature on the subject on identity, mention should be made of the American sociologist and writer Erving Goffman (1922–1982), the creator of the concept of a total institution, i.e. a social organization within which there is a closed group of people who are formally controlled by the staff of this institution [54].

In management sciences, a significant research emphasis is taken in the discourse on organizational identity. The history of transferring the concept of identity to the ground of the community, and thus organization, has a short tradition, because it appeared in the 1980s thanks to the works of Richard Jenkins, who proved that individual and collective

cannot be considered separately, they are inextricably linked [55]. On the other hand, Mary Jo Hatch and M. Schultz describe the differences between organizational identity and corporate identity, as they believe that the former is deeply rooted in the organizational culture with its local meanings and symbols, which are communicated to individual members of the organization by supervisors [54]. They refer to the concept of the American philosopher, sociologist and psychologist George Herbert Mead (1863–1931), proving that the organization has its own identity described as "objective self" and "subjective self" [55]. Researchers Blake E. Ashforth and F. Mael indicate that the process of acquiring social identity is the basic condition for undertaking group activities. The process of identifying with the group is the basis of participating in it. Social identity manifests itself in gaining a sense of the group's separateness, striving to maintain its prestige [56].

According to M. Trotsky, the identity of science can be defined as the awareness of community, encompassing a system of values, individual attitudes, methods of action and goals that determine the unity of the scientific community. In this way, it is possible to perceive the internal identity of a scientific organization, focused on a common research topic, implementing a coherent activity. Its role is to integrate scientists with its environment. In contrast, external identity realizes a sense of separateness in relation to other, foreign scientific environments. The basic concepts within which science functions are three aspects of its understanding: resultant, functional and institutional. When describing the outcome context, science can be defined as the finite result of cognitive activities obtained in the research process. It includes a system of justified hypotheses and theorems subject to scientific cognition, understood according to the best and most up-to-date knowledge. It covers the subject of research, their methodology, and the method of popularization – scientific publications (works, articles in scientific and professional journals). In the functional aspect, we perceive science as a specialized activity aimed at learning and understanding reality, the motto of which is the obligation to use and transfer knowledge to others. Such an under-

standing of the identity of science is diversified due to the scientific discipline, type of research, and even the mode of obtaining degrees and titles as well as the implementation of undergraduate and postgraduate education of students. In the institutional context, science is understood as the activity of all kinds of scientific institutions [57]. They operate on the basis of the classification of scientific fields and disciplines. This is how universities operate - universities, polytechnic universities, medical academies, academies of fine arts, music academies, physical education academies, but also research units, faculties, institutes, departments and departments [58].

Scientific disciplines are often interrelated, both within the same field and even within other fields of science. There may be three types of relationships between individual scientific disciplines: interdisciplinarity, multidisciplinarity and transdisciplinarity [59]. According to the dictionary definition, interdisciplinarity is an interaction between two or more disciplines. It may concern the exchange of ideas, mutual integration in terms of concept perception, methodology, procedures, terminology, and the organization of research and didactics. An interdisciplinary research group is a group of people educated in various disciplines who work on a common research problem through intercommunication [60]. Following J. Axer, it should be assumed that the determinant of interdisciplinarity is the possibility of functioning of groups composed of scientists of different ages, with various competences and views, but undertaking common research problems both in the field of research and didactics. Its success depends on the degree of internal differentiation and the intensity of team cooperation [61]. In the opinion of J. Kurczewska, the foundations of interdisciplinarity are used for specific purposes in order to meet the complexity of scientific research and describe issues on the border of several scientific fields or solve a specific problem that has not yet been explored by researchers of any discipline. We also notice the need to refer to it when we are looking for new conditions for the unity of knowledge [62]. Multidisciplinarity, on the other hand, in the way of defining by W. Gagatka assumes the mutual dependence of two or more

scientific disciplines, while transdisciplinarity means not only communication between researchers of several fields, but above all going beyond their characteristic domains in terms of research [63]. According to M. Goryń, interdisciplinarity can be understood in four ways. The first includes the possibility of supporting research conducted in a given discipline by researchers from disciplines of another field of science. An example of this is the ecology of an organization that brings together researchers in the field of management sciences and biologists. The research interests of organization ecologists focus on the dynamics of the mechanisms of population evolution in the context of relations between the organization and the environment. Population is defined here as a collection of entities with the same characteristics, and the dynamics of change is explained by evolutionary processes, including differentiation, selection and retention. The environment selects those organizations that are best adapted to external conditions, and the population is selected by the environment [64]. Similarly, there are attempts to implement joint activities in the field of pedagogy and medicine, for example in the wide area of health education. Its basic tasks include raising public awareness in the field of disease prevention, for example cancer, which translates directly into the improvement of the health condition of the society. The consequence of cooperation then becomes the emergence of new professions: health educator, health promoter [65]. The second area of interdisciplinarity may assume that research conducted in a given discipline refers to other areas of science. An example of this is researchers who focus on behavioral finance, which is the link between psychology and finance. The research topics include the analysis of individual investor behavior when making specific investment decisions. The apogee of interest in this subject came when the Nobel Prize for work in the field of economics was awarded in 2002 to two psychologists: Daniel Kahneman and Amos Tversky [66], or actually only the first of them, because the second researcher did not live to see the award, dying of cancer in 1996 year. The Kahneman and Tversky model allows for the explanation of the behavior of specialists, including physicians, at the time of their diagnosis [67]. The third do-

main of interdisciplinarity can be considered the area in which research conducted in a given discipline refers to other disciplines from the same field. An example of this is the common research topic undertaken within medical sciences between medicine and public health [68]. Public health is a science dealing with the synthesis of the impact of health systems, human resource management and social awareness in order to obtain a holistic approach to health and disease with a description of their risk factors. In Westerling's definition, it is described in the form of various forms of supervision over the health of the society, including planning and participation in preventive programs, their evaluation, health promotion and undertaking the issues of health care organization [69]. Research undertaken by scientists focused on public health is also of interest to social medicine, preventive medicine, occupational medicine, as well as many medical specialties, both basic and specific. In the fourth area of interdisciplinarity, research conducted in a given sub-discipline can be written, to which those from another sub-discipline refer, however, both remain specified in the scope of the same discipline. An example of cooperation can be research conducted together by a number of medical specialties. The reason for cooperation is then the same problem, for example the disease entity, its understanding, epiopathogenesis, diagnosis and treatment. Here, one has to take into account the necessity to co-solve the problem that is imposed on scientists and doctors with a holistic view of man, which is a challenge for modern medicine. Recently, we all learned the sense and mechanism of this cooperation during the Covid-19 pandemic caused by the SARS-CoV-2 virus infection. We owe the lives of millions of people to the close cooperation of doctors of various specialties: infectious agents, internists, pulmonologists, anesthesiologists, nephrologists, otorhinolaryngologists, cardiologists, etc. In this situation, the issue of interdisciplinarity also appeared at other levels of its examination. Since the outbreak began, efforts to develop a vaccine against the virus have continued throughout the world. Thanks to the multidisciplinary participation in the work of scientists from various research areas and the farthest corners of the world: virologists, doctors, genetic engineers,

pharmacists, economists and even politicians, today we have a chance to overcome the pandemic and return to normalcy.

Summary

The area of management places organizational identity in the group of interdisciplinary issues. Since the beginning of their existence, science schools have been characterized by a strong identification, both in terms of personalities, supported by ties with the scientific authorities creating them, and the issues they undertake. The participants of the science school share similar values that follow the norms and create organizational cultures. It is particularly noticeable in medical disciplines where, from the beginning of the educational and professional path, the master-student relationship is the value and basis for the education of future specialist doctors in their fields. Medical science schools associated with research, publications and scientific authorities with a uniform organizational identity are established in research centers. They become opinion-forming centers which, through their research and educational activities, influence other organizations by taking up the challenge of educating medical staff up to date. It is clearly visible in the system of educating the next generations of specialists, doctors and physiotherapists. Each of them, not only as part of their studies, but also in compulsory postgraduate education, becomes part of the scientific school in which they are educated, and then they transfer their ideas and way of thinking to the organization in which they work, and often even create it from scratch.

The thesis that scientific schools related to research, publication achievements and scientific authorities are established in various medical research centers is confirmed by the examples described in the article. The research thesis is exemplified by the establishment of several medical research centers in Poland with their own identity and organizational culture. It is difficult to say that they are based on various paradigms, but undoubtedly, they have created their own publishing output, specific research fields and scientific authorities.

References

1. Gajda Z. Historia medycyny dla każdego. Warszawa: Wydawnictwo Fronda; 2021, p. 7.

2. Nyczkało N. Problemy mistrzostwa w działalności szkół naukowych: aspekt historyczno- pedagogiczny i perspektywy. In: Bednarczyk H, ed. Filozofia mistrzostwa pedagogicznego. Radom: Instytut Technologii Eksploatacji; 2010, p. 39.

3. K.Opałek K. Zagadnienia teorii prawa i teorii polityki. Warszawa: Państwowe Wydawnictwo Naukowe; 1983, pp. 30–44.

4. Tatarkiewicz W. Historia Filozofii, t. 1. Warszawa: Państwowe Wydawnictwo Naukowe; 1981, pp. 138–145.

5. Popkin RH, Stroll A. Filozofia. Poznań: Wydawnictwo Zysk i Spółka; 1994, pp. 19–26.

6. Muszyński Z. Siedem cech głównych szkoły naukowej. Forum Akademickie 2014; 3(1–2): 63–67.

7. Witczak H. Wstęp do naukowego statusu koncepcji zarządzania. Organizacja i Kierowanie 2014; 2(162): 71–80.

8. Walczak M. Teoria paradygmatu i jej zastosowanie w naukach prawnych. Zeszyty Naukowe Towarzystwa Doktorantów UJ Nauki Społeczne 2015; 10: 71–72.

9. Powszechna Encyklopedia Filozofii. Lublin: Polskie Towarzystwo Tomasza z Akwinu; 2007, p. 86.

10. Kuhn TS. Struktura rewolucji naukowych. Warszawa: Wydawnictwo Aletheia; 2009, p. 54.

11. Rainko S. Dwa paradygmaty. Warszawa: Państwowy Instytut Wydawniczy; 2011, pp. 22–26.

12. Jankowski K. Ewolucja pojęcia paradygmatu w "Strukturze rewolucji naukowych" Thomasa S. Kuhna. ΣΟΦΙΑ. Pismo Filozofów Krajów Słowiańskich 2016; 16: 212–215.

13. Dembiński B. Późna nauka Platona: związki ontologii i matematyki. Katowice: Wydawnictwo Uniwersytetu Śląskiego; 2003, p. 8.

14. Dembiński B. Stara Akademia Platona. W początkach epoki hellenistycznej (ostatni okres). Kęty: Wydawnictwo Marek Derewiecki; 2018, p. 183.

15. Głądczuk Cz. Jak czytać Platona, jak Arystotelesa. Studia Teologiczne 1997; 2: 287–290.

16. Erler M. Platona krytyka pisma/oralności oraz teoria pryncypiów. Peitho. Examina Antiqua 2015; 1(6): 45–47.

17. Czakon W. Szkoły a mody w zarządzaniu strategicznym. Prace naukowe WWSZIP 2014; 27(2): 47–53.

18. Stern M. Greek and Latin Authors on Jews and Judaism. Jerusalem: Isael Academy of Sciences and Humanities, 1976–84. Printed Publications of the Israel Academy of Science and Humanities, pp. 8–12.

19. Tatarkiewicz W. Historia filozofii, t. 1: Filozofia starożytna i średniowieczna. Warszawa: Państwowe Wydawnictwo Naukowe; 1988, p. 18.

20. Iwaszkiewicz PP, Łoś WW, Stępień M. Władcy i wodzowie starożytności. Słownik. Warszawa: WSiP; 1998, p. 119.

21. Łukaszewicz A. Egipt Greków i Rzymian. Warszawa: Książka i Wiedza; 2006, p. 370.

22. Piszczek Z, ed. Mała encyklopedia kultury antycznej. Warszawa: Państwowe Wydawnictwo Naukowe; 1983, p. 182.

23. Kordos M. Wykłady z historii matematyki. Warszawa: Wyd. Script; 2005, pp. 78–86.

24. Kristellen P. Florentine Academy. In: Borchert DM, ed. Encyclopedia of Philosophy. Chicago: Thomson Gale; 2006, pp. 671–672.

25. Kawalec P, Majdański S. Skrypt dla uczestników studiów podyplomowych "Zarządzanie badaniami naukowymi i pracami rozwojowymi w jednostkach naukowych". Lublin: Wydawnictwo Lubelskiej Szkoły Biznesu; 2008, p. 22.

26. Żurawski S. Słowniki tematyczne. Język polski. Nauka o literaturze. Warszawa: Państwowe Wydawnictwo Naukowe; 2011, p. 5.

27. Gołąb-Meyer Z. Galileusz (1564–1642). Foton 2009; 106: 40.

28. Knowles Middleton WE. Science in Rome 1675–1700, and the Accademia Fisicomatematica of Giovanni Giustino Ciampini. British Journal for the History of Science 1975; 8(2): 138–154.

29. Dynastie – Jagiellonowie. Wyd. I. Warszawa: AGORA S.A.; 2010, p. 87.

30. Grzybowski S. Mikołaj Kopernik. Warszawa: Książka i Wiedza; 1972, pp. 72–73.

31. Wroczyński R. Dzieje oświaty polskiej do roku 1795. Warszawa: Państwowe Wydawnictwo Naukowe; 1987, p. 224.

32. Ambroise J. Komisja Edukacji Narodowej w Polsce (1773–1794). Jej dzieło wychowania obywatelskiego. Wrocław–Warszawa–Kraków–Gdańsk: Ossolineum; 1979, p. 278.

33. Chłapowski K. Starostowie niegrodowi w Koronie 1565–1795. Warszawa: Bellerive-Sur-Allier; 2017, p. 86.

34. Więch AS. Polska Nagroda Nobla – mecenat Erazma Józefa Jerzmanowskiego. In: Fac L, ed. Między nauką a oświatą. Na styku szkolnictwa wyższego i średniego w Galicji na przełomie XIX i XX wieku. Przemyśl; 2003, pp. 141–153.

35. Czeżowski T. Kazimierz Twardowski 20 X 1866–11 II 1938. Ruch Filozoficzny 2018; 74(1): 22.

36. Szmyd K. Lwowska Szkoła Filozoficzna w rozwoju nauk o wychowaniu (1914–1939). Współtwórcy, dokonania, kontynuacje. Lubelski Rocznik Pedagogiczny 2016; 35(4): 176.

37. Feliksiak S, ed. Słownik biologów polskich. Warszawa: Państwowe Wydawnictwo Naukowe; 1987, pp. 94–95.

38. Brzęk G, Feliksiak S. Hoyer Henryk Fryderyk. In: Feliksiak S, ed. Słownik biologów polskich. Warszawa: Państwowe Wydawnictwo Naukowe; 1987, pp. 212–214.

39. Zamojski J. Dlaczego wciąż warto badać polską myśl filozoficzno-lekarską. In: Polska szkoła filozofii medycyny. Przedstawiciele i wybrane teksty źródłowe, pod redakcją Michała Musielaka i Jana Zamojskiego. Poznań: Wydawnictwo Naukowe Uniwersytetu Medycznego im. K. Marcinkowskiego w Poznaniu; 2010, pp. 11–15.

40. Brzeziński T. Rozwój nauk medycznych i kształcenia lekarzy w Polsce w XIX i pierwszej połowie XX wieku. In: Brzeziński T, ed. Warszawa: Wyd. Lekarskie PZWL; 2000, p. 456.

41. Bojczuk H., Towarzystwo Medyczne Wileńskie – pierwsze półwiecze (1805–1850). Polska Akademia Nauk, Instytut Historii Nauki. Medycyna Nowożytna 2000; 7(2): 75, 85.

42. Kierzek A. Problem tonsilotomii w ujęciu Teodora Herynga (1847––1925), Jana Sędziaka (1861–1932) i Ludwika Rydygiera (1850––1920). Otolaryngologia Polska 2007; 61(5): 750–757.

43. Kierzek A. Rozwój warszawskiej myśli otolaryngologicznej w XIX wieku. Wrocław: Oficyna Wydawnicza Arboretum; 1997, pp. 77–79.

44. Cichocka-Szumilin I. Polskie Towarzystwo Otolaryngologiczne. In: Słownik Polskich Towarzystw Naukowych, tom I. Wrocław: Wyd. PAN; 1978, pp. 369–372.

45. Gliński J. Słownik biograficzny lekarzy i farmaceutów ofiar drugiej wojny światowej, tom 1. Wrocław: Urban & Partner; 1997, pp. 384–385.

46. Kierzek A. Ćwierćwiecze Sekcji Historycznej Polskiego Towarzystwa Otorynolaryngologów – Chirurgów Głowy i Szyi. Polski Przegląd Otorynolaryngologiczny 2012; 1(2): 161–165.

47. Czaplicki B. Wspomnienia o działalności Sekcji Łódzkiej Polskiego Towarzystwa Oto-Laryngologicznego w okresie 1921–1939, z archiwum dr Anny Pajor.

48. Góralówna M, Kossowska E. Historia otolaryngologii dziecięcej w Polsce. Otolaryngologia Polska 1991; 45, supl.: 325–326.

49. Encyklopedia PWN. Warszawa: Państwowe Wydawnictwo Naukowe; 1987, p. 518.

50. Golka M. Czym bywa tożsamość? Człowiek i Społeczeństwo. Poznań: Uniwersytet im. Adama Mickiewicza w Poznaniu, Wydział Nauk Społecznych; 2006; 26: 9–33.

51. Okoń W. Nowy słownik pedagogiczny. Warszawa: Wydawnictwo Akademickie Żak; 2001: 95.

52. Burdzik T. Przestrzeń jako składnik tożsamości w świecie globalizacji. Kultura–Historia–Globalizacja 2012; 11: 17.

53. Mead GH. Umysł, osobowość, społeczeństwo. Warszawa: Państwowe Wydawnictwa Naukowe; 1975.

54. Goffman E. Człowiek w teatrze życia codziennego, tłum. H. Datner-Śpiewak i P. Śpiewak, oprac. Jerzy Szacki. Wyd I: Warszawa: PIW; Biblioteka Myśli Współczesnej; 1977. Wyd II: Warszawa: Wyd. KR; 2000, p. 286. Wyd III: Warszawa: Wyd. Aletheia; 2008, p. 286.

55. Hańderek J. Kłopoty z tożsamością – kłopoty ze współczesnością. Kwartalnik Filozoficzny 2016; XLIV(2): 5–10.

56. Sułkowski Ł. Kultura jakości w zarządzaniu, czyli pomiędzy tożsamością a kulturą organizacyjną. Przedsiębiorczość i Zarządzanie 2013; XIV(8, cz. II): 25–37.

57. Hatch MJ, Schultz M. The Dynamics of Organisational Identity. In: Hatch MJ, Schultz M, eds. Organisational Identity. A Reader. Oxford–New York: Oxford University Press; 2004, p. 380.

58. Ashforth BE, Mael F. Social Identification Theory and the Organisation. In: Hatch MJ, Schultz M, eds. Organisational Identity. A Reader. Oxford–New York: Oxford University Press; 2004, pp. 134–142.

59. Trocki M. Tożsamość nauk o zarządzaniu. Przegląd Organizacji 2005; 1: 7–8.

60. Gorynia M. Współczesne nauki ekonomiczne: tożsamość, ewolucja, klasyfikacje. Ekonomista 2018; 5, p. 497.

61. Gorynia M. Współczesne nauki ekonomiczne – tożsamość, ewolucja, klasyfikacje. In: Gorynia M, ed., Ewolucja nauk ekonomicznych. Jedność a różnorodność. Relacje do innych nauk. Problemy klasyfikacyjne. Warszawa: Polska Akademia Nauk; 2019, pp. 13–37.

62. Miska-Mazgajczyk E. Nauczanie interdyscyplinarne. Projekt "Twórcza szkoła dla twórczego ucznia" współfinansowany przez Unię Europejską ze środków Europejskiego Funduszu Społecznego w ramach Programu Operacyjnego Kapitał Ludzki Beneficjent projektu – Gmina Wilczyn. Wałcz; 2013, p. 4.

63. Axer J. Interdyscyplinarność i międzydziedzinowość w interesie środowiska akademickiego i otoczenia społecznego uniwersytetu. PAUza Akademicka 2018; 427: 2.

64. Kurczewska J. Wariacje na temat interdyscyplinarności wiedzy. Kraków: Debaty PAU, Głosy w dyskusji; 2011, pp. 80–83.

65. Gagatek W. Dyscyplinarna analiza czy interdyscyplinarna synteza. Uwagi o europeistyce jako kierunku studiów uniwersyteckich. In: Czaputowicz J, ed. Studia europejskie. Wyzwania interdyscyplinarności. Warszawa: Wydawnictwo Uniwersytetu Warszawskiego; 2014, pp. 336–338.

66. Stańczyk S. Sieć w języku ekologów organizacji. Organizacja i Kierowanie 2017; 176(2): 29.

67. Wolska-Prylińska D. Współdziałanie w obszarze profesji społecznych i medycznych. Łódź: Wydawnictwo Uniwersytetu Łódzkiego; 2018, p. 8.

68. Czerwonka M, Gorlewski B. Finanse behawioralne. Warszawa: Oficyna Wydawnicza SGH; 2012, p. 3.

69. Dzik B, Kahneman D, Tversky A. Decyzje 2004; 1: 129–130.

Journal of Health Study and Medicine
2021, No. 2
pp. 105-135
DOI 10.36145/JHSM2021.12

Is Obesity Significantly Connected with Urogenital Disorders? – Systematic Review

Aleksandra Wijata[1]

Joanna Osiak[1]

Kornelia Kędziora-Kornatowska[1]
https://orcid.org/0000-0003-4777-5252

[1] Department and Clinic of Geriatrics, Faculty of Health Sciences,
Nicolaus Copernicus University, Bydgoszcz, Poland

Address for correspondence

Aleksandra Wijata
Department and Clinic of Geriatrics, Faculty of Health Sciences
Nicolaus Copernicus University
13/15 Jagiellońska St., 85-067 Bydgoszcz, Poland
aleksandra_wijata@wp.pl

Abstract

Background: *Obesity was declared an epidemic of the 21st century by WHO. Currently, as many as 39% of adults in the world are overweight and 13% are obese. The number of obese people in the world has tripled since 1975. As is well known, excessive abnormal body weight has a significant impact on the functioning of the entire body. It is very common to emphasize education on cardiovascular implications related to obesity matter – however, overweight and obese patients may also be a group particularly vulnerable to problems related to the urogenital system.*

Objectives: *The aim of the study is to draw attention and discuss the problems related to the above-mentioned system, to which this group of patients is exposed more frequently than patients with normal body weight. They include dysfunctions that do not pose a threat to the patient's life, but significantly reduce the quality of life, such as urinary incontinence and impotence, and severe and even life-threatening diseases such as chronic kidney disease or cancer.*

Material and methods: *Based on numerous data available on this subject and collected from various research centers using Google Scholar and PubMed, the most recent and the most important information on this subject has been summarized. To better present and understand the problem, the mechanisms were also discussed, so that the work is a complete analysis, particularly useful in the current obesity epidemic.*

Results: *Obesity is associated with an increased risk of many diseases of the urogenital system. It causes a worse prognosis and makes treatment less effective. Treatment of obesity should be the first line of treatment of the discussed diseases of the genitourinary system.*

Conclusions: *Obesity is a serious and growing problem all over the world. It has an impact on the epidemiology of many diseases of the urogenital system, therefore paying special attention to its prevention and treatment seems to be one of the most important foundations for the prevention and treatment of dysfunctions of the genitourinary system.*

Key words: *obesity, fertility, uroge*nital, urology

Introduction

Obesity is defined as a complex chronic disease and it is a risk factor for many diseases including cardiovascular diseases, diabetes and cancer, and what is more it has a huge impact on overall health [1, 2]. The prevalence of obesity is increasing year by year and this phenomenon affects not only adults but also children and adolescents [1]. Overweight (including obesity) occurs in over 35% of adults and 11% are obese [3]. The prevalence of pediatric overweight (including obesity) in children aged 5 to 17 years is estimated at about 18%, obesity alone is approximately 2–3% [4, 6]. Obesity is defined as one of the biggest problems of public health [3]. BMI (body mass index) is commonly used as a screening tool to characterize and measure obesity [5]. **BMI can be calculated by dividing your weight (in kilograms) by your height in meters squared** [5]. Overweight is defined as a BMI of 25–30 kg/m^2 and obese is defined as a BMI ≥ 30 kg/m^2, **BMI between 30–34.9 is defined as 1st degree obesity, BMI 35–39.9 2nd degree obesity and over 40 3rd degree obesity** [5].

Mechanism and pathophysiological basis

Major function of ovaries is production, differentiation and release of oocytes. What is more, female gonads produce hormones that influence the development of secondary sex characteristics and the course of pregnancy [7]. One of the most common causes of infertility among women is Polycystic ovary syndrome (PCOS) [8]. PCOS is a reproductive and endocrine disorder and is frequently associated with insulin resistance, metabolic disorders, abdominal adiposity, cardiovascular risk factors and obesity [9]. It affects women of all ages, but most often it occurs in the reproductive age [10]. It is the most common cause of hyperandrogenization and the most common cause of ovulation disorders in women who are treated for infertility [9].

Obesity affects the hypothalamic-pituitary-ovaries (HPO) axis. A normal energy balance, assuming average costs of thermoregulation, reaso-

nable energy consumption and sufficient food consumption, affects the pulsatile secretion of gonadotropin-releasing hormone (GnRH), which translates into the proper functioning of the reproductive axis [11]. GnRH stimulates the pituitary gland to secrete the following hormones: luteinizing hormone (LH) and follicle-stimulating hormone (FSH), which stimulate the growth of follicles in the ovaries and stimulate the production of estradiol and progesterone after ovulation [12, 13]. Estradiol works by feedback and mainly inhibits FSH. On the other hand, when estradiol reaches the concentration threshold and maintains it for at least 24–48 h, it activates increased LH secretion by reducing the amplitude and increasing the frequency of GnRH pulses [14]. Proper maturation is controlled by the hypothalamus and adrenal glands. The adrenal glands take part in adrenarche, which is associated with the increased secretion of androgens of the adrenal cortex in the period preceding puberty or in early puberty [15].

Adipose tissue secretes adipokines, these are bioactive cytokines that are involved in inflammatory processes and metabolic regulation. Due to the secretion of adipokines by adipose tissue, it is considered as an endocrine organ that has an important role in the regulation of physiological events such as immune response, reproduction, glucose and lipid metabolism. The normal level of adipokines is crucial for the functioning of the hypothalamic-pituitary-gonadal axis, as well as for the successful implantation of the embryo, regulation of ovulatory processes and physiological pregnancy. Adipokines specific for adipose tissue include leptin, adiponectin, resin, visfatin and omentin. It has been shown that adipose tissue dysfunction has been observed in patients with PCOS, leading to the overproduction of adipokines. Abnormal concentrations of these factors are strongly correlated with insulin resistance and type 2 diabetes (T2DM) [16].

By affecting the pituitary LH release, obesity leads to a disturbance in the luteal phase. Moreover, it not only affects many other components of the hypothalamic-pituitary-ovarian axis, but may have a direct, independent of the hypothalamus and pituitary effects on the ovaries [17]. Seve-

ral changes in the inflammatory and metabolic systems associated with ovarian dysfunction are outlined below.

Leptin

Leptin is one of the adipokines which, by signaling at the level of the hypothalamus, suppresses appetite and stimulates energy expenditure. Leptin was investigated as a potential slimming drug, but it turned out that the increased concentration of exogenous leptin did not cause weight loss [18]. This study suggested leptin resistance at the hypothalamus level. Another study showed a high concentration of leptin in obese women, which confirms the potential resistance to leptin [19].

Leptin also influences maturation, as proper energy balance is required for proper puberty [20]. Moreover, hyperleptinemia may initiate premature reproductive maturation, while the prolonged state of high leptin concentration may lead to abnormal ovulation [21]. On the other hand, low leptin concentration in the physiological period of puberty may delay or lack puberty [22].

Leptin affects the pulsatile secretion of GnRH; its non-physiological concentration is associated with delayed maturation and reduced fertility [23].

The optimal concentration of leptin is necessary for the proper functioning of the HPO axis, its low concentration may disrupt the physiological secretion of GnRh, on the other hand, high levels may disrupt ovarian folliculogenesis [21].

Ghrelin

Ghrelin is an enterokinase produced mainly by the stomach, but it is also detected in the pancreas, intestine, pituitary and hypothalamus [24]. Its concentration is increased on an empty stomach, and it stimulates the appetite to cover low nutrient intake [25].

In response to nutritional changes, ghrelin influences the homeostasis of reproductive function, which may reduce GnRH secretion and pulsation. In states of low nutrient intake, the concentration of ghrelin

increases, which inhibits the activity of HPO [26]. Moreover, ghrelin influences the secretion of other pituitary hormones, for example: adrenocorticotropic hormone and prolactin. Low levels of ghrelin in low energy states can stimulate the secretion of prolactin, potentially leading to disturbances in ovarian cyclicity. There was also a decrease in ghrelin concentration in children as people approached puberty [27].

In the case of obesity, ghrelin concentration decreases with increasing body weight in the pubertal period [28].

Neuropeptide Y and Agouti related protein (NPY/AgRP)

The arcuate nucleus, which secretes NPY and AGRP, connects peripherally circulating enterokines, adipokines, and the HPO axis. NPY is a neuropeptide that stimulates fat proliferation and angiogenesis, while AgRp stimulates the appetite. Both interact with ghrelin and stimulate the appetite [29].

Under physiological conditions, NPY concentration increases during the ovulatory release to enhance the effect of GnRH, which stimulates the secretion of FSH and LH from the pituitary gland [28]. On the other hand, AgRP stimulates the hypothalamus to generate GnRH pulses, which regulates the release of gonadotropins [30]. NPY may have antiproliferative and pro-apoptotic effects on ovarian folliculogenesis [31].

Adiponectin

Adiponectin is an adipokine secreted by adipose tissue and its concentration increases with hunger [32]. It works by binding to the receptor in the arcuate nucleus, which leads to a reduction in energy expenditure and higher food consumption [33]. Moreover, the production of adiponectin makes cells more sensitive to insulin, and its concentration is inversely correlated with obesity [34]. Low levels of adiponectin are associated with insulin resistance, type 2 diabetes and metabolic syndrome [35].

TNF-α, IL-6 and IL-8 are pro-inflammatory factors that contribute to the development of insulin resistance [36]. Adiponectin inhibits the pro-

duction of TNF-α in adipocytes by acting as an anti-inflammatory adipokine [34]. In obese women, pro-inflammatory factors are elevated, while the concentration of adiponectin is low [21].

Adiponectin receptors can also be found in the ovaries. During the preovulatory phase, adiponectin is involved in changes in the cells of the granular layer, interacting with insulin and IGF-1 [37].

Insulin

Insulin is produced in the beta cells of the pancreas, and glucose stimulates its release. Insulin concentration increases with obesity, and insulin sensitivity decreases [37]. Insulin synthesis is stimulated by adipose tissue. Hyperinsulinemia affects the liver, reducing the production of SHBG, leading to an increase in the concentration of free circulating steroid hormones – estrogens and androgens [38]. The effect of LH on the ovaries is enhanced by insulin, which increases the secretion and production of androgens [39]. Additionally, excessive LH secretion may inhibit follicle growth in the early stages, which promotes cell luteinization and, consequently, may reduce the quality of oocytes [43].

By acting on the pituitary gland, insulin stimulates GnRH, which in turn causes the release of LH [40]. Insulin increases FSH activity by stimulating ovarian steroidogenesis [41]. In genetic diseases, a relationship between the syndromic form of insulin resistance and gonadotropin-independent hyperandrogenism and ovarian enlargement is observed [42].

The state of insulin resistance in particular affects the muscles, liver and adipose tissue, while the ovaries remain insulin sensitive and are therefore exposed to the effects of hyperinsulinemia. Insulin stimulates the cells of the ovaries to produce androgens, and their excess may lead to premature follicular atresia, and thus anovulation [43].

Hyperinsulinemia and insulin resistance accompanying obesity in women may cause disturbances in ovulation, menstruation and fertility.

Obesity affects the reproductive apparatus by impeding the development of ovarian follicles, quantitative and qualitative disorders of oocyte maturation, and disturbs fertilization, meiosis and the appropriate

preimplantation of the embryo [44]. The high concentration of free fatty acids has a toxic effect on reproductive tissues, which contributes to chronic inflammation and cell damage. The excess fatty acids supplied to the body with food can be stored in the form of triglycerides in the adipocytes, which does not cause cell damage. On the other hand, in the case of adipocyte overload, fatty acids accumulate in other tissues, which causes a toxic effect called lipotoxicity [45].

Obesity in the male population

Obesity affects the reproductive functions of men in a multifactorial way and is associated with reduced fertility [46, 47]. In overweight and obese men, an increased probability of decreased fertility and abnormal sperm parameters was observed [48].

Oligozoospermia and low ejaculation volume were more frequent in men with increased waist circumference (WC) and BMI. **The study showed that in men WC > 101.6 cm there is a 7 times greater chance of oligozoospermia, and obese men (BMI > 30) had a 19 times greater chance of oligozoospermia** [49]. There was also a correlation between obesity in men and a higher percentage of abnormal sperm and a lower sperm concentration [50]. Moreover, studies of men undergoing fertility treatment with assisted reproductive technology (ART) showed a relationship between high BMI and a decrease in the pregnancy rate and the number of live births [51]. Despite extensive knowledge of obesity and infertility in women, the impact of obesity in men on the reproductive system is not fully understood.

It has been suggested that male infertility is influenced by several mechanisms that are not yet fully understood and elucidated. The negative impact of obesity on male fertility is mainly associated with a lower serum testosterone level and an increased concentration of estradiol, and therefore spermatogenesis is impaired. Moreover, obesity, through atherosclerotic influence on blood vessels, leads to erectile dysfunction. Increased inflammation and increased testicular temperature are also responsible for the disorders of spermatogenesis [52]. Normally in men,

the hypothalamus produces and releases gonadotropin releasing hormone (GnRH), which stimulates the anterior pituitary gland to produce and release luteinizing hormone (LH) and follicle stimulating hormone (FSH). LH and FSH then stimulate the nucleus to stimulate steroidogenesis and spermatogenesis. Obese men have normal or decreased serum LH levels, decreased total testosterone levels and decreased SHBG levels [53].

Obesity and the associated sedentary lifestyle cause an increase in testicular temperature, which contributes to the increase in estrogen production and disrupts the HPG axis [54]. Adipose tissue produces adipokines that contribute to inflammation and oxidative stress, which can contribute to damage to the structures of the testicles and epididymis. Testosterone production seems to be regulated by leptin, and in obese men a correlation has been shown between high leptin levels and hypogonadism [53]. It should be remembered that fertility is also influenced by other obesity-related factors, such as hyperlipidemia, pro-inflammatory states, metabolic syndrome and cardiovascular diseases [55].

Male obesity may disrupt the molecular and physical structure of sperm during spermatogenesis in the testicle, but also during sperm maturation in the epididymis. People with a high BMI may have a reduced semen quality, which is associated with a lower concentration and sperm motility, as well as a reduced acrosome response and a higher risk of DNA damage [53].

With the global obesity epidemic, there has been a sharp increase in male infertility. This phenomenon is mainly related to the imbalance of sex hormones, abnormal production of inflammatory factors and harmful effects on spermatogenesis [53].

Nephrolithiasis

Obesity and overweight are very significant risk factors for many diseases of the urinary system. Patients with an increased BMI show, among others, an increased tendency to develop kidney diseases,

problems with urinary incontinence and it can also cause growth in cancer incidence.

Urolithiasis is one of the most common urinary tract ailments. Stones in the urinary system may be a random find in an imaging examination (usually during ultrasonography) or may cause symptoms, most often in the form of renal colic. In the United States, urolithiasis affects about 8.8% of the population. It is more common in men than in women and was more often found among obese people (11.2%) compared to people with normal body weight (6.1%) [56]. The obesity epidemic observed in recent years and the change in eating habits mean that excessive body weight and the occurrence of metabolic syndrome can be considered a factor associated with the occurrence of urolithiasis. Urine tests in overweight people have shown that poor eating habits that lead to overweight and obesity may cause changes in urine biochemistry and because of that more kidney stones in these people [57].

There are likely three mechanisms by which obesity can be linked to an increased incidence of nephrolithiasis. The main mechanism is related to the fact that obesity is often associated with insulin resistance, which together lead to a low urine pH and thus uric acid precipitation and the formation of urate stones. The second mechanism is an increased amount of lithogenesis promoters and a decreased amount of its inhibitors in obese people. The last factor is improper diet often used by obese people. There was also a positive correlation between BMI and the amount of urinary oxalate, uric acid, sodium and phosphorus excreted in the urine [58].

Urinary incontinence (UI)

Another problem that obese patients may face more often than the general population is stress urinary incontinence [59]. Population studies have shown that UI is more common in women than in men, and that about 10% of all adult women suffer from it [60].

The cause of stress urinary incontinence is a loss of support for the pelvic floor muscles and connective tissue. The etiology of this disorder is multifactorial and includes, but is not limited to, connective tissue di-

sorders, chronic cough, obesity, pelvic floor injuries after vaginal delivery, pregnancy, menopause, constipation, weightlifting and smoking [61].

In the treatment of stress incontinence, one of the first recommendations is weight loss, giving up smoking and urorehabilitation. Studies have shown that weight loss intervention reduced the frequency of stress urinary incontinence episodes for 12 months and improved patient satisfaction with changes in urinary incontinence for 18 months [62]. Based on the results obtained, it may be suggested that maintaining body weight may have long-term benefits in the treatment of urinary incontinence.

Other renal dysfunctions

The diet of overweight and obese patients is often based on eating large amounts of carbohydrates and fats (especially saturated ones). As it turns out, the type of food you eat can have a direct impact on kidney damage. One study found that a long-term high-fat diet causes at least some kidney damage due to tissue lipid accumulation, increased oxidative stress, and mitochondrial dysfunction that promote excessive programmed cell death [63]. This may be associated with a greater incidence of various renal dysfunction, including chronic kidney disease [64]. The major physiological responses of the kidneys to obesity include increased glomerular filtration rate, renal plasma flow, filtration fraction and tubular sodium reabsorption. Considering obesity as a risk factor for kidney disease, obesity-related glomerulopathy (ORG) was distinguished, as a disease connected with excess body weight. The incidence of ORG is increasing in parallel with the worldwide obesity epidemic. The most common symptom is subnephrotic proteinuria in the urinalysis [65]. The important is that studies have shown that weight loss led to a reduction in proteinuria in patients with ORG [66].

Infections

Numerous studies have examined whether obesity is associated with an increased incidence of urinary tract infections. Many of them show that overweight people are at greater risk of developing **UTI (urinary tract**

infection) and pyelonephritis [67]. A similar dependence was observed in children [68].

The important fact is that adipose tissue is also actively involved in inflammation and immunity. Leptin is also involved in these mechanisms. A genetic defect with leptin deficiency in mice, which causes a severe obesity phenotype, is associated with impaired phagocytic functions and a decrease in T-lymphocyte function. In these mice, this increases the susceptibility to bacterial infections, e.g., Listeria monocytogenes, Klebsiella pneumoniae, etc. Leptin deficiency is associated with susceptibility for infections in both animals and humans [69].

Recurrent urinary tract infections may be associated with other causes, therefore in such patients, despite excessive body weight, in-depth diagnostics should be considered.

Renal cell cancer (RCC)

Overweight and obesity strongly correlate with the incidence of kidney cancer. Meta-analyzes suggest that overweight / obesity increases the risk of kidney cancer in both men and women [70]. Abdominal obesity can also be a risk factor in kidney cancer.

The mechanism of this dependency has not been fully explained. Also, as in the case of kidney stones, the presence of kidney tumors is associated with hyperinsulinemia. Insulin resistance, which is common in obesity and leads to an increase in IGF-1 levels. IGF-1 plays a role in carcinogenesis and leads to an increased risk of cancer [71].

An interesting phenomenon related to excess body weight and kidney cancer is the so-called obesity paradox. The obesity paradox is based on the observation that obesity is a risk factor for developing kidney cancer, but obese patients have an increased survival compared to normal BMI. However, the biological mechanisms underlying this observation are not yet fully understood. Recently, aspects of the tumor microenvironment that vary with BMI in tumor and peritumoral adipose tissue have been discovered, which may contribute to the apparent survival advantage in obese patients [72].

Diagnostics

According to the WHO, overweight in adults is diagnosed if the BMI value is within the range of 25–30 kg/m^2, while the diagnosis of obesity is based on a BMI ≥ 30 kg/m^2. Adults should be screened annually for overweight or obesity using BMI calculations. Please note that BMI is not an ideal indicator. In athletes, the elderly and the sick as well as the disabled (especially physically disabled) and people with sarcopenia, BMI is not an appropriate anthropometric indicator to assess the presence of excessive amounts of adipose tissue. The reason for this is the different ratio of muscle and fat mass in these people compared to a statistically healthy person. Based on the International Diabetes Federation (IDF) diagnostic criteria in adult Europeans with a waist circumference greater than (or equal to) 94 in men and 80 in women, visceral obesity can be diagnosed.

Patients with excessively abnormal body weight should be under the constant supervision of family doctors. Since an obese patient has an increased risk of diseases of the genitourinary system, the presence of this factor should be considered when reporting symptoms and informing the patient about the importance of weight loss at every stage of the diagnosis. One of the first and most important diagnostic tools that can guide us to the correct diagnosis is medical record.

A meta-analysis of 10 studies confirmed that women with a positive clinical history (presence of coughing, sneezing, walking or running as initiators of an incontinence episode) had a 74% chance of stress urinary incontinence, while women with a negative clinical history had a 34% chance [73]. There are also international standardized questionnaires that are routinely used in urinary incontinence clinical trials, including Incontinence, Impact Questionnaire (IIQ), King's Health Questionnaire (KHQ) and Urogenital Distress Inventory (UDI) [74]. In addition to the history and questionnaires, the initial evaluation includes physical examination, micturition diary, general urine examination, postvoidal residual urine assessment in ultrasound, and a pad test. Diagnostics is based mainly on the initial evaluation and in some patients on urodynamic tests

[75]. Doctors should not perform cystoscopy on healthy women who are considering surgery to evaluate stress urinary incontinence unless there are concerns about abnormalities in the urinary tract [76].

In the event of alarm symptoms such as haematuria, a tumor palpable through the abdominal wall, a positive family history, pain in the lumbar region, fever, and refractory arterial hypertension, the diagnosis should always be significantly deepened. A patient with unexplained hematuria should always be referred to a urologist for cystoscopy. There are no specific guidelines for the diagnosis of a patient with excess body weight. However, it should be remembered that excess body weight is a factor that increases the risk of both chronic kidney disease and urinary tract cancer. It is also important to inform at every stage of the diagnosis how important weight loss is. Weight reduction in obesity not only has a nephroprotective effect [77] but it is one of the main recommendations in the treatment of urinary incontinence.

Obesity treatment

One of the ways of losing weight is diet therapy, which assumes achieving a net energy deficit [78]. Obtaining a caloric deficit is possible in several ways, including by appropriate composition of macronutrients, limiting calories, changing meals [79].

In addition to a reduced calorie diet and exercise, drug treatment of obesity is recommended.

Orlistat

Orlistat acts on pancreatic lipases by irreversibly inhibiting their action, which reduces the absorption of free fatty acids and their excretion in the faeces. Orlistat reduces BMI, body weight, waist circumference, and cholesterol levels. Better glycemic control has been shown in people with diabetes. The most common side effect is gastrointestinal disturbance, which is often the reason why therapy is discontinued [80].

Liraglutide

Liraglutide is an agonist at the GLP-1 (glucagon-like peptide-1) receptor. GLP-1 is a released incretin hormone that is released from the gastrointestinal tract in response to the ingestion of glucose and fat. It works peripherally, by slowing down the passage of the digestive tract, and centrally, suppressing the appetite [81].

Naltrexone/bupropion

Naltrexone is an opioid antagonist used to treat opioid and alcohol dependence. Bupropion inhibits the uptake of norepinephrine and dopamine and is used as an adjunct to quitting therapy. When combined as a combined drug, they reduce appetite [82].

When other obesity treatments have failed, bariatric surgery is the treatment of choice [83]. Bariatric surgery is recommended for patients who meet the following criteria:

- BMI ≥ 40 kg/m^2
- BMI ≥ 35 kg/m^2 with associated comorbidities that could be improved with weight loss
- BMI of 30–34.9 kg/m^2 who have recent-onset T2DM
- other weight loss options have been explored but have failed
- patient is receiving or will receive intensive management in a tier 3 service (a service-based weight loss program)
- patient is fit for anaesthesia and surgery propos
- patient shows commitment to long-term follow-up [84].

Proposed methods of bariatric treatment:

- **Adjustable Laparoscopic Gastric Banding** – is placed around the upper part of the stomach. The band narrows the stomach's lumen, limiting the amount of food that can be swallowed.
- **Roux-en-Y gastric bypass** – involves the simultaneous reduction of the stomach and intestinal absorption, thus enabling effective weight reduction. The procedure begins with the creation of a small stomach just below the cardia, i.e., connecting the stomach with the esophagus. The intestine is then divided into two parts

– the lower (Roux loop), which is then pulled up and attached to the new stomach, and the higher (enzyme loop), which is attached to the distal end of the Roux loop. From now on, gastric juices and digestive enzymes will flow through the enzyme loop to the Roux loop, where they will combine with the food and digestion will begin there.

- **Sleeve gastrectomy** – during the operation, 80% of the stomach is removed, while the 'sleeve' is left, in the narrow medial part. The reduced size of the stomach causes a reduction in the volume of food consumed.
- **Biliopancreatic diversion with a duodenal switch.**
- **Revision and reversal of bariatric surgery** [84].

Discussion

This article aims to emphasize the importance of obesity in the development of disorders of the genitourinary system, which not only increases morbidity and mortality, but also reduces the quality of life. Given that obesity rates are increasing worldwide, especially among women of childbearing age, this creates not only a clinical problem, but also a financial problem in health care [85]. That is why primary prevention and obesity prevention are so important, which will relieve health care. Moreover, the results of studies on increased fertility after weight loss are controversial, increasing the importance of primary prevention and promoting a healthy lifestyle [86]. Moreover, studies conducted on a group of men who underwent bariatric surgery showed a decrease in the number of already disturbed sperm. On the other hand, sperm parameters of men treated with diet and exercise improved [87].

Increased hemodynamics and the metabolic burden associated with obesity aggravate chronic kidney disease that has already developed [88]. What is important – non-surgical weight loss interventions reduce proteinuria and blood pressure and appear to prevent further deterioration of renal function [89]. Nevertheless, most patients believe that their

excess weight adversely affects their health and are aware of its consequences. They consider losing weight to be too difficult. Patients often mention the lack of motivation or money as a problem [90]. Therefore, it is most important that weight loss should be the mainstay of treatment of urogenital disorders in patients with excess body weight. Since health problems are a less motivating aspect for young adults than for middle-aged **adults** [91], the increased emphasis on primary prevention in young people seems to be the most effective idea, while reducing costs for the treatment of obesity complications.

Conclusions

Obesity and overweight are a disease of civilization, which in women of childbearing age may have a significant impact on their fertility, and thus may cause suffering due to the lower likelihood of pregnancy. The chronic inflammatory state of obesity can directly affect the normal function of the ovaries. Energy balance is closely related to the reproductive system and is controlled by many hormones: adipokines, cytokines and growth factors. The effect on reproductive function disorders occurs through neuroendocrine mechanisms that interfere with the functions of the ovaries and proper ovulation. Endocrine disorders, mainly decreased levels of gonadotropins and estradiol, also have a negative effect on reduced fertility. In turn, decreased fertility in obese men results from many genetic, physical and hormonal mechanisms that lead to abnormal sperm parameters and erectile dysfunction.

References

1. Włodarczyk M, Nowicka G. Obesity, DNA Damage, and Development of Obesity-Related Diseases. Int J Mol Sci 2019 Mar 6; 20(5): 1146. https://doi: 10.3390/ijms20051146. PMID: 30845725; PMCID: PMC6429223.

2. Żukiewicz-Sobczak W, Wróblewska P, Zwoliński J, Chmielewska-Badora J, Adamczuk P, Krasowska E, Zagórski J, Oniszczuk A, Piątek J, Silny W. Obesity, Diabetes, and Cardiovascular Diseases: A Compendium. Annals of Agricultural and Environmental Medicine 2014; 21(3): 590–594.

3. Żukiewicz-Sobczak W, Wróblewska P, Zwoliński J, Chmielewska-Badora J, Adamczuk P, Krasowska E, Zagórski J, Oniszczuk A, Piątek J, Silny W. Obesity and poverty paradox in developed countries. Ann Agric Environ Med 2014; 21(3): 590–594. https://doi: 10.5604/12321966.1120608. PMID: 25292135.

4. Oude Luttikhuis H, Baur L, Jansen H, Shrewsbury VA, O'Malley C, Stolk RP, Summerbell CD. WITHDRAWN: Interventions for treating obesity in children. Cochrane Database Syst Rev 2019; 3(3): CD001872. https://doi: 10.1002/14651858.CD001872.pub3.

5. Tremmel M, Gerdtham U-G, Nilsson PM, Saha S. Economic Burden of Obesity: A Systematic Literature Review. Int J Environ Res Public Health 2017; 14: 435.

6. Goldsammler M, Merhi Z, Buyuk E. Role of hormonal and inflammatory alterations in obesity-related reproductive dysfunction at the level of the hypothalamic-pituitary-ovarian axis. Reprod Biol Endocrinol 2018; 16(1): 45.

7. Vurbic D, Harder VS, Redner RR, Lopez AA, Phillips JK, Higgins ST. Co-occurring obesity and smoking among U.S. women of reproductive

age: Associations with educational attainment and health biomarkers and outcomes. Prev Med 2015; 80: 60–66. https://doi:10.1016/j.ypmed.2015.05.020.

8. Cena H, Chiovato L, Nappi RE. Obesity, Polycystic Ovary Syndrome, and Infertility: A New Avenue for GLP-1 Receptor Agonists. J Clin Endocrinol Metab 2020 Aug 1; 105(8): e2695–709. https://doi: 10.1210/clinem/dgaa285. PMID: 32442310; PMCID: PMC7457958. Escobar-Morreale HF. Polycystic ovary syndrome: definition, aetiology, diagnosis and treatment. Nature Reviews Endocrinology 2018.

9. McGee EA, Hsueh AJ. Initial and cyclic recruitment of ovarian follicles. Endocr Rev 2000 Apr; 21(2): 200–214. https://doi: 10.1210/edrv.21.2.0394. PMID: 10782364.

10. Michalakis K, Mintziori G, Kaprara A, Tarlatzis BC, Goulis DG. The complex interaction between obesity, metabolic syndrome and reproductive axis: a narrative review. Metabolism 2013; 62(4): 457–478.

11. Belchetz PE, Plant TM, Nakai Y, Keogh EJ, Knobil E. Hypophysial responses to continuous and intermittent delivery of hypothalamic gonadotropin-releasing hormone. Science 1978 Nov 10; 202(4368): 631–633. https://doi: 10.1126/science.100883. PMID: 100883.

12. Adashi EY. Endocrinology of the ovary. Hum Reprod 1994 May; 9(5): 815–827. https://doi: 10.1093/oxfordjournals.humrep.a138602. PMID: 7929728.

13. Evans NP, Dahl GE, Mauger DT, Padmanabhan V, Thrun LA, Karsch FJ. Does estradiol induce the preovulatory gonadotropin-releasing hormone (GnRH) surge in the ewe by inducing a progressive change in the mode of operation of the GnRH neurosecretory system. Endocrinology 1995 Dec; 136(12): 5511–5519. https://doi: 10.1210/endo.136.12.7588302.

14. Counts DR, Pescovitz OH, Barnes KM, Hench KD, Chrousos GP, Sherins RJ, Comite F, Loriaux DL, Cutler GB Jr. Dissociation of adrenarche and gonadarche in precocious puberty and in isolated hypogonadotropic hypogonadism. J Clin Endocrinol Metab 1987 Jun; 64(6): 1174–1178. https://doi: 10.1210/jcem-64-6-1174. PMID: 3571422.

15. Silvestris E, de Pergola G, Rosania R, Loverro G. Obesity as disruptor of the female fertility. Reprod Biol Endocrinol 2018 Mar 9; 16(1): 22. https://doi: 10.1186/s12958-018-0336-z. PMID: 29523133; PMCID: PMC5845358.

16. Jain A, Polotsky AJ, Rochester D, Berga SL, Loucks T, Zeitlian G, Gibbs K, Polotsky HN, Feng S, Isaac B, Santoro N. Pulsatile luteinizing hormone amplitude and progesterone metabolite excretion are reduced in obese women. J Clin Endocrinol Metab 2007 Jul; 92(7): 2468–2473. https://doi: 10.1210/jc.2006-2274. Epub 2007 Apr 17. PMID: 17440019.

17. Bozkurt L, Göbl CS, Rami-Merhar B, Winhofer Y, Baumgartner-Parzer S, Schober E, Kautzky-Willer A. The Cross-Link between Adipokines, Insulin Resistance and Obesity in Offspring of Diabetic Pregnancies. Horm Res Paediatr 2016; 86: 300–308. https://doi: 10.1159/000448076.

18. Considine RV, Sinha MK, Heiman ML, Kriauciunas A, Stephens TW, Nyce MR, Ohannesian JP, Marco CC, McKee LJ, Bauer TL et al. Serum immunoreactive-leptin concentrations in normal-weight and obese humans. N Engl J Med 1996 Feb 1; 334(5): 292–295. https://doi: 10.1056/NEJM199602013340503. PMID: 8532024.

19. Frisch RE, Revelle R. Height and weight at menarche and a hypothesis of critical body weights and adolescent events. Science 1970 Jul 24; 169(3943): 397–399. https://doi: 10.1126/science.169.3943.397. PMID: 5450378.

20. Gosman GG, Katcher HI, Legro RS. Obesity and the role of gut and adipose hormones in female reproduction. Hum Reprod Update 2006 Sep–Oct; 12(5): 585–601. https://doi: 10.1093/humupd/dml024. Epub 2006 Jun 14. PMID: 16775192.

21. Egan OK, Inglis MA, Anderson GM. Leptin Signaling in AgRP Neurons Modulates Puberty Onset and Adult Fertility in Mice. J Neurosci 2017 Apr 5; 37(14): 3875–3886. https://doi: 10.1523/JNEUROSCI.3138-16.2017. Epub 2017 Mar 8. PMID: 28275162; PMCID: PMC6596709.

22. Zuure WA, Roberts AL, Quennell JH, Anderson GM. Leptin signaling in GABA neurons, but not glutamate neurons, is required for reproductive function. J Neurosci 2013; 33(45): 17874–17883. https://doi: 10.1523/JNEUROSCI.2278-13.2013.

23. Tena-Sempere M. Roles of ghrelin and leptin in the control of reproductive function. Neuroendocrinology 2007; 86(3): 229–241. https://doi: 10.1159/000108410. Epub 2007 Sep 12. PMID: 17851226.

24. Asakawa A, Inui A, Kaga T, Yuzuriha H, Nagata T, Ueno N, Makino S, Fujimiya M, Niijima A, Fujino MA, Kasuga M. Ghrelin is an appetite-stimulatory signal from stomach with structural resemblance to motilin. Gastroenterology 2001 Feb; 120(2): 337–345. https://doi: 10.1053/gast.2001.22158. PMID: 11159873.

25. Nicolas R. Vulliémoz, Ennian Xiao, Linna Xia-Zhang, Marc Germond, Jean Rivier, Michel Ferin. Decrease in Luteinizing Hormone Pulse Frequency during a Five-Hour Peripheral Ghrelin Infusion in the Ovariectomized Rhesus Monkey. The Journal of Clinical Endocrinology & Metabolism 2004 1 Nov; 89(11): 5718–5723.

26. Broglio F, Benso A, Castiglioni C, Gottero C, Prodam F, Destefanis S, Gauna C, van der Lely AJ, Deghenghi R, Bo M, Arvat E, Ghigo E. The endo-

crine response to ghrelin as a function of gender in humans in young and elderly subjects. J Clin Endocrinol Metab 2003; 88(4): 1537–1542.

27. Whatmore AJ, Hall CM, Jones J, Westwood M, Clayton PE. Ghrelin concentrations in healthy children and adolescents. Clin Endocrinol (Oxf) 2003 Nov; 59(5): 649–654. https://doi: 10.1046/j.1365--2265.2003.01903.x. PMID: 14616891.

28. Cansell C, Denis RG, Joly-Amado A, Castel J, Luquet S. Arcuate AgRP neurons and the regulation of energy balance. Front Endocrinol (Lausanne) 2012; 3: 169. https://doi:10.3389/fendo.2012.00169.

29. Kuo LE, Kitlinska JB, Tilan JU, Li L, Baker SB, Johnson MD, Lee EW, Burnett MS, Fricke ST, Kvetnansky R, Herzog H, Zukowska Z. Neuropeptide Y acts directly in the periphery on fat tissue and mediates stress-induced obesity and metabolic syndrome. Nat Med 2007; 13(7): 803–811.

30. Sirotkin AV, Kardošová D, Alwasel SH, Harrath AH. Neuropeptide Y directly affects ovarian cell proliferation and apoptosis. Reprod Biol 2015 Dec; 15(4): 257–260. https://doi: 10.1016/j.repbio.2015.07.004. Epub 2015 Aug 4. PMID: 26679167.

31. Lee B, Shao J. Adiponectin and energy homeostasis. Rev Endocr Metab Disord 2014 Jun; 15(2): 149–156. https://doi: 10.1007/s11154-013-9283-3. PMID: 24170312; PMCID: PMC4006341.

32. Kubota N, Yano W, Kubota T, Yamauchi T, Itoh S, Kumagai H, Kozono H, Takamoto I, Okamoto S, Shiuchi T, Suzuki R, Satoh H, Tsuchida A, Moroi M, Sugi K, Noda T, Ebinuma H, Ueta Y, Kondo T, Araki E, Ezaki O, Nagai R, Tobe K, Terauchi Y, Ueki K, Minokoshi Y, Kadowaki T. Adiponectin stimulates AMP-activated protein kinase in the hypothalamus and increases food intake. Cell Metab 2007 Jul; 6(1): 55–68. https://doi: 10.1016/j.cmet.2007.06.003. PMID: 17618856.

33. Maeda N, Shimomura I, Kishida K, Nishizawa H, Matsuda M, Nagaretani H, Furuyama N, Kondo H, Takahashi M, Arita Y, Komuro R, Ouchi N, Kihara S, Tochino Y, Okutomi K, Horie M, Takeda S, Aoyama T, Funahashi T, Matsuzawa Y. Diet-induced insulin resistance in mice lacking adiponectin/ACRP30. Nat Med 2002 Jul; 8(7): 731–717.

34. Kadowaki T, Yamauchi T, Kubota N, Hara K, Ueki K, Tobe K. Adiponectin and adiponectin receptors in insulin resistance, diabetes, and the metabolic syndrome. J Clin Invest 2006 Jul; 116(7): 1784–1792. https://doi: 10.1172/JCI29126. PMID: 16823476; PMCID: PMC1483172.

35. Thaler JP, Schwartz MW. Minireview: Inflammation and obesity pathogenesis: the hypothalamus heats up. Endocrinology 2010 Sep; 151(9): 4109–4115. https://doi: 10.1210/en.2010-0336. Epub 2010 Jun 23. PMID: 20573720; PMCID: PMC2940486.

36. Chabrolle C, Tosca L, Dupont J. Regulation of adiponectin and its receptors in rat ovary by human chorionic gonadotrophin treatment and potential involvement of adiponectin in granulosa cell steroidogenesis. Reproduction 2007 Apr; 133(4): 719–731.

37. Lundon JR. Insulin-some current concepts. Can Fam Physician 1970 Jul; 16(7): 58–61. PMID: 20468537; PMCID: PMC2281770.

38. Preziosi P, Barrett-Connor E, Papoz L, Roger M, Saint-Paul M, Nahoul K, Simon D; Interrelation between plasma sex hormone-binding globulin and plasma insulin in healthy adult women: the telecom study. J Clin Endocrinol Metab 1993 Feb; 76(2): 283–287.

39. Brothers KJ, Wu S, DiVall SA, Messmer MR, Kahn CR, Miller RS, Radovick S, Wondisford FE, Wolfe A. Rescue of obesity-induced infertility in female mice due to a pituitary-specific knockout of the insulin receptor. Cell Metab 2010 Sep 8; 12(3): 295–305. https://doi: 10.1016/j.cmet.2010.06.010. PMID: 20816095; PMCID: PMC2935812.

40. Willis D, Mason H, Gilling-Smith C, Franks S. Modulation by insulin of follicle-stimulating hormone and luteinizing hormone actions in human granulosa cells of normal and polycystic ovaries. J Clin Endocrinol Metab 1996 Jan; 81(1): 302–309.

41. Ou XH, Li S, Wang ZB, Li M, Quan S, Xing F, Guo L, Chao SB, Chen Z, Liang XW, Hou Y, Schatten H, Sun QY. Maternal insulin resistance causes oxidative stress and mitochondrial dysfunction in mouse oocytes. Hum Reprod 2012 Jul.

42. Poretsky L, Cataldo NA, Rosenwaks Z, Giudice LC. The insulin-related ovarian regulatory system in health and disease. Endocr Rev 1999 Aug; 20(4): 535–582. https://doi: 10.1210/edrv.20.4.0374. PMID: 10453357.

43. Qiao J, Feng HL. Extra- and intra-ovarian factors in polycystic ovary syndrome: impact on oocyte maturation and embryo developmental competence. Hum Reprod Update 2011; 17(1): 17–33. https://doi: 10.1093/humupd/dmq032.

44. Jungheim ES, Travieso JL, Hopeman MM. Weighing the impact of obesity on female reproductive function and fertility. Nutr Rev 2013 Oct; 71 Suppl 1(0 1): S3–8. https://doi: 10.1111/nure.12056.

45. Sørensen TI, Virtue S, Vidal-Puig A. Obesity as a clinical and public health problem: is there a need for a new definition based on lipotoxicity effects? Biochim Biophys Acta 2010 Mar; 1801(3): 400–404. https://doi: 10.1016/j.bbalip.2009.12.011.

46. Chambers TJ, Richard RA. The impact of obesity on male fertility. Hormones (Athens) 2015 Oct–Dec; 14(4): 563–568. https://doi: 10.14310/horm.2002.1621. PMID: 26732149.

47. Sundaram R, Mumford SL, Buck Louis GM. Couples' body composition and time-to-pregnancy. Hum Reprod 2017 Mar 1; 32(3): 662–668. https://doi: 10.1093/humrep/dex001. PMID: 28158570; PMCID: PMC5400044.

48. Campbell JM, Lane M, Owens JA, Bakos HW. Paternal obesity negatively affects male fertility and assisted reproduction outcomes: a systematic review and meta-analysis. Reprod Biomed Online 2015 Nov; 31(5): 593–604. https://doi: 10.1016/j.rbmo.2015.07.012. Epub 2015 Aug 10. PMID: 26380863.

49. Eisenberg ML, Kim S, Chen Z, Sundaram R, Schisterman EF, Buck Louis GM. The relationship between male BMI and waist circumference on semen quality: data from the LIFE study. Hum Reprod 2014 Feb; 29(2): 193–200. https://doi: 10.1093/humrep/det428.

50. Tsao CW, Liu CY, Chou YC, Cha TL, Chen SC, Hsu CY. Exploration of the association between obesity and semen quality in a 7630 male population. PLoS One 2015 Mar 30; 10(3): e0119458. https://doi: 10.1371/journal.pone.0119458. PMID: 25822490; PMCID: PMC4379020.

51. Mushtaq R, Pundir J, Achilli C, Naji O, Khalaf Y, El-Toukhy T. Effect of male body mass index on assisted reproduction treatment outcome: an updated systematic review and meta-analysis. Reprod Biomed Online 2018 Apr 36(4): 459–471. https://doi: 10.1016/j.rbmo.2018.01.002.

52. Katib A. Mechanisms linking obesity to male infertility. Cent European J Urol 2015; 68(1): 79–85. https://doi: 10.5173/ceju.2015.01.435. Epub 2015 Mar 13. PMID: 25914843; PMCID: PMC4408383.

53. Liu Y, Ding Z. Obesity, a serious etiologic factor for male subfertility in modern society. Reproduction 2017 Oct; 154(4): R123–R131. https://doi: 10.1530/REP-17-0161.

54. Best D, Avenell A, Bhattacharya S. How effective are weight-loss interventions for improving fertility in women and men who are overweight or obese? A systematic review and meta-analysis of the evidence. Hum Reprod Update 2017 Nov 1; 23(6): 681–705. https://doi: 10.1093/humupd/dmx027.

55. McPherson NO, Lane M. Male obesity and subfertility, is it really about increased adiposity? Asian J Androl 2015; 17(3): 450–458. https://doi: 10.4103/1008-682X.148076.

56. Charles D. Scales Jr. Prevalence of Kidney Stones in the United States. European Urology 2012; 62(1): 160–165.

57. Shavit L, Ferraro PM, Johri N, Robertson W, Walsh SB, Moochhala S, Unwin R. Effect of being overweight on urinary metabolic risk factors for kidney stone formation. Nephrol Dial Transplant 2015 Apr; 30(4): 607–613. https://doi: 10.1093/ndt/gfu350. Epub 2014 Oct 31. PMID: 25362001.

58. Brynarski P. Cukrzyca i otyłość jako czynniki ryzyka wystąpienia kamicy nerkowej. www.mp.pl [Internet]. Available from https://diabetologia.mp.pl/powiklania/inne/205979,cukrzyca-i-otylosc-jako-czynniki-ryzyka-wystapienia-kamicy-nerkowej [cited 15.03.2019].

59. Lamerton TJ, Torquati L, Brown WJ. Overweight and obesity as major, modifiable risk factors for urinary incontinence in young to mid-aged women: a systematic review and meta-analysis. Obes Rev 2018 Dec; 19(12): 1735–1745. https://doi: 10.1111/obr.12756. Epub 2018 Sep 19. PMID: 30230164.

60. Milsom I, Gyhagen M. The prevalence of urinary incontinence. Climacteric 2019 Jun; 22(3): 217–222. https://doi: 10.1080/13697137.2018.1543263.

61. Lugo T, Riggs J. Stress Incontinence [Updated 2020 Nov 20]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2021 Jan. Available from: https://www.ncbi.nlm.nih.gov/books/NBK539769/.

62. Wing RR, West DS, Grady D, Creasman JM, Richter HE, Myers D, Burgio KL, Franklin F, Gorin AA, Vittinghoff E, Macer J, Kusek JW, Subak LL. Program to Reduce Incontinence by Diet and Exercise Group. Effect of weight loss on urinary incontinence in overweight and obese women: results at 12 and 18 months. J Urol 2010 Sep; 184(3): 1005–1010. https://doi: 10.1016/j.juro.2010.05.031. PMID: 20643425; PMCID: PMC3038435.

63. Sun Y, Ge X, Li X, He J, Wei X, Du J, Sun J, Li X, Xun Z, Liu W, Zhang H, Wang ZY, Li YC. High-fat diet promotes renal injury by inducing oxidative stress and mitochondrial dysfunction. Cell Death Dis 2020 Oct 24; 11(10): 914. https://doi: 10.1038/s41419-020-03122-4. PMID: 33099578; PMCID: PMC7585574.

64. Ross WR, McGill JB. Epidemiology of obesity and chronic kidney disease. Adv Chronic Kidney Dis 2006 Oct; 13(4): 325–335. https://doi: 10.1053/j.ackd.2006.07.012. PMID: 17045219.

65. D'Agati, VD, Chagnac A, de Vries APJ, Levi M, Porrini E, Herman-Edelstein M, Praga M. Obesity-related glomerulopathy: clinical and pathologic characteristics and pathogenesis. Nat Rev Nephrol 2016; 12(8): 453–471. https://doi.org/10.1038/nrneph.2016.75.

66. Shen WW, Chen HM, Chen H, Xu F, Li LS, Liu ZH. Obesity-related glomerulopathy: body mass index and proteinuria. Clin J Am Soc Nephrol 2010 Aug; 5(8): 1401–1409. https://doi: 10.2215/CJN.01370210. Epub 2010 May 24. PMID: 20498244; PMCID: PMC2924407.

67. Semins MJ, Shore AD, Makary MA, Weiner J, Matlaga BR. The Impact of Obesity on Urinary Tract Infection Risk. Urology 2012 Feb; 79(2): 266–269.

68. Hsu Po-Chang, Chen Shyi-Jou. Obesity and risk of urinary tract infection in young children presenting with fever. Medicine 2018 Dec; 97(49): e13006. https://doi: 10.1097/MD.0000000000013006.

69. Matthew E Falagas, Maria Kompoti. Obesity and infection. Lancet Infect Dis 2006; 6: 438–446.

70. Liu Xuezhen, Qi Sun, Haifeng Hou, Kai Zhu, Qian Wang, Huamin Liu, Qianqian Zhang, Long Ji, Dong Li. The association between BMI and kidney cancer risk: An updated dose – response meta – analysis in accordance with PRISMA guideline. Medicine 2018 Nov; 97(44): e12860. https://doi:10.1097/MD.0000000000012860.

71. Nam Ga Eun, Cho Kyung Hwan, Han Kyungdo, Kim Chul Min et al. Obesity, abdominal obesity and subsequent risk of kidney cancer: a cohort study of 23.3 million East Asians. Br J Cancer 2019; 121: 271–277. https://doi.org/10.1038/s41416-019-0500-z.

72. Sanchez A. et al. Transcriptomic signatures related to the obesity paradox in patients with clear cell renal cell carcinoma: a cohort study. Lancet Oncol 2020 Feb; 21(2): 283–293. https://doi.org/10.1016/S1470-2045(19)30797-1.

73. Holroyd-Leduc JM, Tannenbaum C, Thorpe KE et al: What type of urinary incontinence does this woman have? JAMA 2008; 299(12): 1446–1456.

74. Bender S, Borowski J, Borkowski T, Torz C, Radziszewski P. Nietrzymanie moczu. Medycyna po Dyplomie 2011(20); 6(183): 73–80.

75. Drewa T, Juszczak K. Urologia. Ilustrowany podręcznik dla studentów i stażystów. Warszawa: PZWL; 2018.

76. Kobashi KC, Albo ME, Dmochowski RR et al. Surgical Treatment of Female Stress Urinary Incontinence: AUA/SUFU Guideline. J Urol 2017 Oct; 198(4): 875.

77. Paulina Smektała, Paweł Bogdański. Nephroprotective role of body mass reduction in obesity. Forum Zaburzeń Metabolicznych 2011; 2(3): 198–204.

78. Hall KD, Sacks G, Chandramohan D, Chow CC, Wang YC, Gortmaker SL, Swinburn BA. Quantification of the effect of energy imbalance on bodyweight. Lancet 2011 Aug 27; 378(9793): 826–837. https://doi: 10.1016/S0140-6736(11)60812-X. PMID: 21872751; PMCID: PMC3880593.

79. Nakamura T, Kuranuki S. Nutrition – Macronutrients. In: Vasan RS, Sawyer DB, eds. Encyclopedia of cardiovascular research and medicine. Elsevier; 2018.

80. Agam B. Bansal, Yasir Al Khalili. Orlistat In: StatPearls. Treasure Island (FL): StatPearls Publishing; 2021 Jan.

81. Gadde KM, Martin CK, Berthoud HR, Heymsfield SB. Obesity: Pathophysiology and Management. J Am Coll Cardiol 2018; 71(1): 69–84. https://doi: 10.1016/j.jacc.2017.11.011.

82. National Institute for Health and Care Excellence Technology appraisal guidance: Naltrexone – bupropion for managing overweight and obesity. Technology appraisal guidance [TA494]. NICE; 2014 [Internet]. Available from: https://www.nice.org.uk/guidance/ta494/resources/naltrexonebupropion-for-managing-overweight-and-obesity-pdf-82605086955205.

83. Colquitt JL, Pickett K, Loveman E, Frampton GK. Surgery for weight loss in adults. Cochrane Database Syst Rev 2014; 8(8): CD003641.

84. Ruban A, Stoenchev K, Ashrafian H, Teare J. Current treatments for obesity. Clin Med (Lond) 2019; 19(3): 205–212. https://doi:10.7861/clinmedicine.19-3-205.

85. Chandrasekaran S, Neal-Perry G. Long-term consequences of obesity on female fertility and the health of the offspring. Curr Opin Obstet Gynecol 2017; 29(3): 180–187.

86. Amiri M, Ramezani Tehrani F. Potential Adverse Effects of Female and Male Obesity on Fertility: A Narrative Review. Int J Endocrinol Metab 2020; 18(3): e101776. Published 2020 Sep 28. https://doi:10.5812/ijem.101776.

87. Chambers TJ, Richard RA. The impact of obesity on male fertility. Hormones (Athens) 2015; 14(4): 563–568.

88. Snyder S, Turner GA, Turner A. Obesity-related kidney disease. Prim Care 2014 Dec; 41(4): 875–893.

89. Navaneethan SD, Yehnert H, Moustarah F, Schreiber MJ, Schauer PR, Beddhu S. Weight loss interventions in chronic kidney disease: a systematic review and meta-analysis. Clin J Am Soc Nephrol 2009; 4(10): 1565–1574. https://doi: 10.2215/CJN.02250409. Epub 2009 Sep 17. PMID: 19808241; PMCID: PMC2758256.

90. Saeed Z, Janda KM, Tucker BM, Dudley L, Cutter P, Friedman AN. Personal Attitudes Toward Weight in Overweight and Obese US Hemodialysis Patients. J Ren Nutr 2017 Sep; 27(5): 340–345. https://doi: 10.1053/j.jrn.2017.03.004. Epub 2017 May 19. PMID: 28533101.

91. Lanoye A, Grenga A, Leahey TM, LaRose JG. Motivation for weight loss and association with outcomes in a lifestyle intervention: comparing emerging adults to middle aged adults. Obes Sci Pract 2018; 5(1): 15–20. https://doi: 10.1002/osp4.313.