COVID-19 Influence on Newborns

Yusupova M.A., Abdirimova A.D.
Urgench State Medical Institute, Urgench, Uzbekistan

The new coronavirus infection caused by the SARS-CoV2 virus has become a global problem for humanity incredibly quickly due to its high contagiousness and mortality among the population. An extremely unfavorable epidemiological situation has developed worldwide, which has led to high morbidity among pregnant women [2,9]. During the pandemic, maternal and perinatal morbidity and mortality rates associated with the new COVID-19 coronavirus infection and its complications increased significantly, demonstrating the unpreparedness of medical workers worldwide to combat the new disease due to insufficient knowledge of its pathogenesis and treatment possibilities [1,17,18,24,32,41]. During the pandemic, data were accumulated on the peculiarities of the course of the COVID-19 infection, the clinical manifestations of which are variable: from asymptomatic course to the development of massive lung damage and acute respiratory distress syndrome. Due to mutations in the SARS-CoV-2 virus strain, the disease can be unpredictable and cause complications in people of any age. During the COVID-19 pandemic, several significant increases in morbidity related to SARS-CoV-2 virus mutations were noted, characterized by variability in clinical and laboratory data and mortality rates [2,9,19,25,34,40].

This article analyzes observations and assesses perinatal outcomes in women with SARS-CoV-2 associated infection, comparing them with data from publications on COVID-19.

The infection caused by the SARS-CoV-2 virus, detected in a pregnant woman in the first trimester, did not lead to an increase in the thickness of the fetal collar zone, the development of congenital anomalies, or delayed development, and did not increase the risk of miscarriage compared to other transplacental infections, such as the Zika virus [7,8]. At the same time, clinical observations of pregnancies against the background of SARS-CoV-2 infection in later stages, ending in premature birth and even perinatal fetal death, resulting in maternal mortality [8], have been described. It is not fully understood whether premature termination of pregnancy is a complication

of this infection or is caused by obstetric tactics aimed at early delivery of sick women. Further research is needed to determine the impact of the new coronavirus infection on pregnancy, the placenta, and related complications in the fetus [3,10,20,26,33,42].

Characteristics of newborns from mothers who have had COVID-19 during pregnancy. In most literature sources devoted to the analysis of the course of the new coronavirus infection in pregnant women, women in labor, and newborns, the somatic status and morphofunctional characteristics of children born to women with a positive PCR test for COVID-19 at the time of delivery were assessed [3, 6]. Considering the hypercoagulation observed in COVID-19 patients, perinatal complications in children are likely due to impaired fetoplacental perfusion and/or possible thrombotic changes in the mother, vasculopathy, decreased placental barrier function, and inflammatory changes in it [5, 6]. D. Baud et al. suggested that virus-induced changes in the placenta lead to chronic and acute fetal hypoxia, premature birth, which determines the severity of the children's condition at birth [1,7].

According to a multi-center cohort study conducted from March 1 to May 10, 2020, in New York City, which included 149 mother-child couples hospitalized in the observation departments, 12% of newborns required hospitalization in the intensive care unit, of which 10% were born prematurely and 3% required artificial lung ventilation. It should be noted that children born to mothers with clinical manifestations of COVID-19 were more often born prematurely (compared to 3% in the general population) and required intensive care (compared to 2%) [4,11,16,27,31,39].

A systematic review published in 2020 compared maternal and neonatal outcomes in women infected with SARS-CoV, SARS-CoV-2, and MERS. Compared to SARS-CoV and MERS, women's SARS-CoV-2 infection was also associated with a higher rate of premature birth and surgical delivery through cesarean section, as well as the frequency of fetal distress syndrome diagnosis and perinatal mortality rates [7].

S. Reem et al. analyzed data on the health status of 201 newborns: 71 children (35.3%) were born prematurely, before 36 weeks of pregnancy. There is no reliable data on the reasons for premature birth: were it obstetric indications or the mother's new coronavirus infection. The average score on the Apgar scale in the 1st minute was

6.49, in the 5th minute - 8.98. In 7 children (2.8%), fetal distress was observed, which in some cases was an indication for preterm labor. After birth, the frequency of respiratory disorders among newborns was 4.4%: 6 newborns had transient neonatal tachypnea, 2 had pneumonia according to radiography, and 1 child had respiratory distress syndrome (RDS). In the sample under consideration, 5 (2.5%) deaths were registered, which is significantly higher than the average statistical indicators (2 cases of stillbirth, 3 more died shortly after birth due to progressive multiple organ failure against the background of infectious-toxic shock). All newborns underwent antibacterial therapy [5,12,15,28,35,38].

H. Zhu et al. reported that out of 10 newborns born to 9 women with COVID-19, 6 were born prematurely, 6 out of 10 developed fetal distress, 6 out of 10 were diagnosed with RDS, 2 had thrombocytopenia, and all children had a negative PCR test for COVID-19 [4].

Several studies have noted the presence of SARS-CoV-2 in placenta, umbilical cord, and amniotic fluid samples, however, the virus's impact on the mother and fetus is currently unclear [7].

It has been noted that in pregnancies that occurred against the background of SARS and MERS, intrauterine fetal developmental delay (IFD) is often recorded [25]. The frequency of POI in pregnancies against the background of a new coronavirus infection did not exceed the general population data [20].

Transmission of the SARS-CoV-2 virus to newborns. The possibility of SARS-CoV-2 transmission from mother to fetus and from mother to newborn is an important issue during the COVID-19 pandemic. To date, there is no consensus on whether the SARS-CoV-2 virus can be transmitted from the mother to the newborn [7]. There is no convincing evidence to claim that the placental barrier reliably prevents the vertical transmission of SARS-CoV-2 [9]. Previous studies on SARS and MERS-induced diseases have not reported cases of their vertical transmission [30, 31]. Similar results were obtained in the first studies of pregnant women infected with SARS-CoV-2, although later studies did not rule out the possibility of intrauterine transmission of the virus to the fetus [6,13,21,23,30,37]. Several authors have published data on SARS-

CoV-2 penetration through the placenta and cases of possible intrauterine SARS-CoV-2 transmission [2, 3].</2

The impact of the SARS-CoV-2 virus on the embryo and/or fetus in the first and second trimester of pregnancy has been little studied. It has been proven that in the placenta of women who have had COVID-19 in the third trimester, maternal/fetal vascular anomalies (malperfusion) develop more frequently [5], and in some clinical observations, the vertical transmission of SARS-CoV-2 has been demonstrated [2, 3]. Moreover, the detection of immunoglobulin M (IgM) against SARS-CoV-2 in the umbilical cord blood and/or in the blood of newborns confirms suspicion of an intrauterine infection, as IgM is not transmitted transplacentally from the mother to the fetus [3]. Detection of SARS-CoV-2 RNA in placental and amniotic tissue samples indicates the possibility of fetal infection during childbirth [4]. Based on histological examination data, placental infection (inflammatory infiltrate in the subchorial space, increased fibrin deposition in the intervillous space, and funisitis) was identified, which also indicates the presence of an inflammatory reaction in the fetus [6, 7].

A.J. Vivanti et al. described a clinical case of SARS-CoV-2 transplacental transmission in a 23-year-old pregnant woman with COVID-19 infection. At birth, SARS-CoV-2 was identified by PCR in amniotic fluid samples, placental tissue, mother's and newborn's blood, and in a nasopharyngeal swab. The viral load in the placental tissues was significantly higher than in the amniotic fluid and maternal blood. Immunohistochemical examination of placental tissue also revealed a high level of SARS-CoV-2 invasion into trophoblast cells and inflammatory changes in the placenta [7,8,14,22,29,36]. The realization of congenital infection in a newborn confirms transplacental transmission in the case of high viral load in placental tissues and the presence of SARS-CoV-2 in trophoblast cells [3,11]. A team of authors from Italy described 2 newborns with a positive PCR result for SARS-CoV-2, obtained immediately after birth. In both cases, SARS-CoV-2 was observed to invade the fetal tissue of the placenta [2].

H. Hosier et al. reported on a case where SARS-CoV-2 was identified in the syncytiotrophoblast of the placenta, although the results of lung, heart, liver, and

kidney analysis of the fetus were negative for SARS-CoV-2 [2]. M. Zamaniyan, A. Ebadi et al. reported that amniotic fluid obtained during cesarean section in a pregnant woman with COVID-19 was positive for SARS-CoV-2 based on PCR results [4]. On the contrary, H. Chen et al. showed that 6 of the 6 amniotic fluid and umbilical cord blood samples tested for SARS-CoV-2 in pregnant women with symptoms and confirmed COVID-19 infection were negative for SARS-CoV-2 [5]. Similar work conducted by Simöes E. Silva et al. demonstrated that the SARS-CoV-2 virus was absent in 18 of the 18 amniotic fluid, cord, and/or placenta samples during 5 different studies. Additionally, the same group of researchers found that 85 (95.5%) of 89 newborns had a negative PCR smear from the nasopharynx for SARS-CoV-2, performed in the first 9 days after birth from mothers with COVID-19, indicating a low probability of intrauterine SARS-CoV-2 transmission. Considering that 4 out of 5 newborns with a positive SARS-CoV-2 result had smears taken 3-9 days after birth, and 3 out of 4 newborns had close contact with infected mothers from the first days, SARS-CoV-2 infection is likely the result of postnatal transmission [3].

Transplacental transmission of antibodies. A newborn's protection from infection depends primarily on innate immunity and maternal antibodies obtained transplacentally. Assessing the level of maternal antibodies produced in response to an infection caused by the SARS-CoV-2 coronavirus during pregnancy, penetrating the placenta, is necessary to understand the potential protection of newborns from COVID-19. Passive immunization during breastfeeding (BF) is also important for child protection. However, data on the level of AT to SARS-CoV-2 in pregnant women who have had COVID-19 and their newborns are not so extensive.

In a prospective observational study conducted by scientists at the Stanford University of the USA, involving 147 newborns from 145 women infected with COVID-19 during pregnancy, IgG levels in maternal blood samples taken during childbirth and in umbilical cord blood were strongly correlated. High transplacental IgG transfer rates were observed in cases where infection occurred less than 60 days before delivery or during the second trimester of pregnancy. The preservation of

maternal IgG in newborns positively correlated with the initial level of antibodies in the umbilical cord blood [6].

The study, conducted by a group of scientists from Germany led by K. Rathberger, included 16 women infected with SARS-CoV-2 during pregnancy and their newborns. The SARS-CoV-2 antibody response was measured in the mother's blood and umbilical cord blood, as well as in the postpartum period and 6-11 weeks after childbirth. A total of 73% of women and 1/3 of their children developed antibodies to SARS-CoV-2. At the same time, the long interval between infection and childbirth, as well as the high titer of maternal antibodies, favorably influenced the transplacental transfer of AT to the fetus. In all children, during subsequent examination, a decrease in antibody titer was observed, while titers in their mothers remained stable or even increased [7]. Post-vaccination immune response is still poorly understood. According to a group of Israeli authors, vaccination with BNT162b2 in the third trimester of pregnancy causes a strong humoral immune response in the mother (IgG antibodies to the S-protein region, called RBD antigen). Antibodies penetrate the transplacental barrier and reach the maternal titers in the fetus within 15 days after vaccination with the first dose of the vaccine [11,12]. The ratio of maternal and neonatal antibodies to COVID-19 did not differ compared to the previous infection. The transmission coefficient of IgG at birth was significantly lower during infection in the third trimester compared to the second trimester [8]. Thus, transplacental transfer of SARS-CoV-2-specific antibodies is possible. However, immunity acquired by newborns transplacentally can be unstable. non-pregnant women belong to the high-risk group for severe NCI, therefore, the issue of timely diagnosis of disease progression, development of somatic, gestational, and perinatal complications is acute, the pathophysiological prerequisites of which lie in both the pregnant woman's body's reactivity to SARS-CoV-2 and the patterns of uncomplicated pregnancy formation. Disruption of gestational adaptation due to NCI leads to a pronounced escalation of physiological shifts (activation of the endothelial-platelet link, moderate pro-inflammatory and hypercoagulation state, regulatory oxidative stress, etc.) with transformation into pathogenetic links of COVID-19 (antiangiogenic, inflammatory

and prothrombogenic status, pronounced systemic and intracellular oxidative stress, hyperactivation of innate immune and metabolic processes with the implementation of PON).

References

1. Ibodullaev, B. B. (2021). Structure and correction of psychoemothical disorders in patients with second type of diabetes. *Asian Journal of Multidimensional Research*, *10*(9), 227-230.

2. Muxiddin, A., & Malika, M. (2016). Epidermal growth factor and its role in the diagnosis polypoid rhinosinusitis. *European research*, (3 (14)), 92-96.

3. Niyazmetov, B., & Avezov, M. (2025). JANUBIY OROL BO'YI MINTQASIDA KO'Z KASALLIKLARINING TURLI YOSHDAGILAR ORASIDA TARQALGANLIGINING XUSUSIYATLARINI O'RGANISH. *SOUTH ARAL SEA MEDICAL JOURNAL*, *1*(4), 749-753.

4. Niyazmetov, B., & Avezov, M. (2025). KO'Z KASALLIKLARINING TURLI YOSHDAGI ODAMLARI ORASIDA TARQALISHI, NOGIRONLIKKA CHIQISH DARAJASINI O'RGANISH. *SOUTH ARAL SEA MEDICAL JOURNAL*, *1*(4), 745-748.

5. Ollaberganova, R. Z., & Ibodullaev, B. B. (2024). Clinical structure and psychocorrection of psychoemotional disorders in patients with hypothyroidism. *American Journal of Medicine and Medical Sciences*, *14*(2), 278-281.

6. Авезов, М. И. (2017). ЭФФЕКТИВНОСТЬ ЛАЗЕРОТЕРАПИИ В ЛЕЧЕНИИ ПОЛИПОЗНОГО ЭТМОИДИТА. *МОЛОДЫЕ УЧЕНЫЕ–МЕДИЦИНЕ*, 5.

7. Авезов, М. И. ДИАГНОСТИЧЕСКАЯ ЦЕННОСТЬ БЕЛКА Р53 ПРИ ПОЛИПОЗНОМ РИНОСИНУСИТЕ. *МЕЖДИСЦИПЛИНАРНЫЙ ПОДХОД ПО ЗАБОЛЕВАНИЯМ ОРГАНОВ ГОЛОВЫ И ШЕИ*, 326.

8. Авезов, М. И., & Джаббаров, К. Д. ДИАГНОСТИЧЕСКАЯ ЦЕННОСТЬ БЕЛКА Р53, ЭПИДЕРМАЛЬНОГО ФАКТОРА РОСТА И ЭНДОТЕЛИАЛЬНОГО ФАКТОРА РОСТА СОСУДОВ (VEGF) ПРИ

ПОЛИПОЗНОМ РИНОСИНУСИТЕ. *ЎЗБЕКИСТОН РЕСПУБЛИКАСИ ОТОРИНОЛАРИНГОЛОГЛАРНИНГ IУ СЪЕЗДИГА БАҒИШЛАНГАН МАҲСУС СОН*, 96.

9. Авезов, М. И., & Ўрунова, Ю. А. (2024). СЕНСОНЕВРАЛ ЭШИТИШ ПАСТЛИГИНИ ДАВОЛАШ САМАРАДОРЛИГИНИ ТАКОМИЛЛАШТИРИШ. *AMALIY VA TIBBIYOT FANLARI ILMIY JURNALI*, *3*(12), 319-321.

10. Авезов, М. И., & Ўрунова, Ю. А. (2024). СЕНСОНЕВРАЛ ЭШИТИШ ПАСТЛИГИНИ ДАВОЛАШ САМАРАДОРЛИГИНИ ТАКОМИЛЛАШТИРИШ. *AMALIY VA TIBBIYOT FANLARI ILMIY JURNALI*, *3*(12), 319-321.

11. Авезов, М. И., Мадаминова, М. Ш., & Садуллаева, А. Ф. Epidermal growth factor and its role in the diagnosis polypoid rhinosinusitis Avezov M., Madaminova M. 2, Sadullayeva A. 3 (Republic of Uzbekistan) Эпидермальный фактор роста и его роль в диагностике полипозного риносинусита. *EUROPEAN RESEARCH: INNOVATION IN SCIENCE, EDUCATION AND TECHNOLOGY*, 92.

12. Авезов, М. И., Рахимов, А. П., & Юсупов, Д. Д. РОЛЬ ОНКОМАРКЕРОВ В ДИАГНОСТИКЕ ПОЛИПОЗНОГО РИНОСИНУСИТА (ОБЗОР ЛИТЕРАТУРЫ). *КОЛОНКА РЕДАКТОРА*.

13. Аллаярова, С. Д., & Юсупова, М. А. (2017). Дифференциально-диагностические критерии обструктивного бронхита, ассоциированного с различными инфекциями респираторного тракта у детей. In *Научный диалог: Молодой ученый* (pp. 10-12).

14. Аллаярова, С. Д., & Юсупова, М. А. (2018). Акушерские причины развития сепсиса и септического шока. *Авиценна*, (21), 12-16.

15. Базарбаева, Д. А., & Юсупова, М. А. (2019). Современные методы диагностики и лечения миомы матки у женщин репродуктивного возраста. *Вестник науки и образования*, (22-3 (76)), 56-59.

16. Базарбаева, Д. А., & Юсупова, М. А. (2020). Хронический эндометрит: современные аспекты диагностики и клинические критерии. *Проблемы современной науки и образования*, (8 (153)), 48-52.

17. Базарбаева, Д. А., & Юсупова, М. А. (2020). Экстракорпоральное оплодотворение в лечении бесплодия. In *НАУЧНЫЕ ДОСТИЖЕНИЯ СТУДЕНТОВ И УЧАЩИХСЯ* (pp. 121-123).

18. Джаббаров, К. Д., & Авезов, М. И. (2014). ДИАГНОСТИЧЕСКАЯ ЦЕННОСТЬ БЕЛКА P53, ЭПИДЕРМАЛЬНОГО ФАКТОРА РОСТА И ЭНДОТЕЛИАЛЬНОГО ФАКТОРА РОСТА СОСУДОВ (VEGF) ПРИ ПОЛИПОЗНОМ РИНОСИНУСИТЕ. *Евразийский журнал здравоохранения*, *3*(3 (1)), 145-149.

19. Джаббаров, К. Д., Рузметов, У. У., Расулов, А. Б., & Авезов, М. И. (2014). ЭФФЕКТИВНОСТЬ ПРЕПАРАТА ФУЗАФУНГИНА В КОМПЛЕКСНОЙ ТЕРАПИИ ОСТРЫХ РИНОСИНУСИТОВ У БЕРЕМЕННЫХ. *Евразийский журнал здравоохранения*, *3*(3 (1)), 55-57.

20. Джуманиязов, С. С., & Юсупова, М. А. (2017). ВНЕБОЛЬНИЧНАЯ ПНЕВМОНИЯ БЕРЕМЕННЫХ: ОСОБЕННОСТИ ЭТИОПАТОГЕНЕЗА И КЛИНИЧЕСКОЙ КАРТИНЫ. *МОЛОДЫЕ УЧЕНЫЕ–МЕДИЦИНЕ*, 81.

21. Джуманиязов, С. С., & Юсупова, М. А. (2017). ОСОБЕННОСТИ ГЕМОСТАЗИОЛОГИЧЕСКИХ ПОКАЗАТЕЛЕЙ У БЕРЕМЕННЫХ СТЯЖЕЛОЙ ВНЕБОЛЬНИЧНОЙ ПНЕВМОНИЕЙ. In *Проблемы гемостаза в хирургии XXI века* (pp. 21-26).

22. Джуманязов, С. С., & Юсупова, М. А. (2019). К вопросу об изменении иммунного статуса у женщин с невынашиванием беременности. *Авиценна*, (28), 10-13.

23. Джуманязов, С. С., & Юсупова, М. А. (2019). КЛИНИКО-ДИАГНОСТИЧЕСКИЕ АСПЕКТЫГАСТРОДУОДЕНАЛЬНОЙ ПАТОЛОГИИ У ДЕТЕЙ С ДИСПЛАЗИЕЙ СОЕДИНИТЕЛЬНОЙ ТКАНИ. In *ПРОРЫВНЫЕ НАУЧНЫЕ ИССЛЕДОВАНИЯ: ПРОБЛЕМЫ, ЗАКОНОМЕРНОСТИ, ПЕРСПЕКТИВЫ* (pp. 333-336).

24. Ибадуллаев, Б. Б. (2022). РОЛЬ ПСИХОТЕРАПИИ В КОРРЕКЦИИ ПСИХОЭМОЦИОНАЛЬНЫХ РАССТРОЙСТВ У БОЛЬНЫХ ДИАБЕТОМ II ТИПА. *Вестник фундаментальной и клинической медицины*, (4), 4.

25. Ибадуллаев, Б. Б., Курбонбоев, С. К., & Сетиризаев, И. Б. (2025). ПОСТХОЛЕСИСТЕКТОМИК СИНДРОМ РИВОЖЛАНГАН БЕМОРЛАРДА ПСИХОЭМОЦИОНАЛ БУЗИЛИШЛАРНИ КОРРЕКЦИЯ ҚИЛИШДА ТИББИЙ-ПСИХОЛОГИК ЁНДАШУВ. *Modern digital technologies in education: problems and prospects*, *2*(5), 26-27.

26. Ибадуллаев, Б. Б., Рахимбаев, М. Д., Каршиев, З. Х., & Нураддинова, М. У. ХАРАКТЕРИСТИКА УРОВНЯ ПСИХОЭМОЦИОНАЛЬНЫХ РАССТРОЙСТВ У БОЛЬНЫХ ДИАБЕТОМ II ТИПА И МЕДИКО-ПСИХОЛОГИЧЕСКИЙ ПОДХОД К ИХ КОРРЕКЦИИ. *J Neural Transm*, *116*, 777-784.

27. Ибадуллаев, Б. Б., Сетиризаев, И. Б., & Курбонбоев, С. К. (2025). СУРУНКАЛИ ГАСТРИТ БИЛАН КАСАЛЛАНГАН БЕМОРЛАРДА АДАПТАЦИОН БУЗИЛИШЛАР КЛИНИК СРУКТУРАСИ ВА ПСИХОКОРРЕКЦИЯСИ. *Modern digital technologies in education: problems and prospects*, *2*(5), 24-25.

28. Ибодуллаев, Б. Б. (2021). ҚАНДЛИ ДИАБЕТ ИККИНЧИ ТИПИ БИЛАН КАСАЛЛАНГАН БЕМОРЛАРДА РИВОЖЛАНГАН ҲАВОТИРЛИ БУЗИЛИШЛАР ПСИХОКОРРЕКЦИЯСИ. *Academic research in educational sciences*, *2*(6), 1107-1112.

29. Ибрагимова, Н. Ш., & Юсупова, М. А. (2020). Факторы риска венозных тромбоэмболических осложнений в онкохирургии. *Авиценна*, (66), 17-19.

30. Каримов, Р., & Авезов, М. (2021). Оценка перинатальных случаев смерти, уровня и состояния заболеваний уха, горла и носа. *Журнал вестник врача*, *1*(1), 60-63.

31. Курбанов, С. Д., & Юсупова, М. А. (2004). Клинико-лабораторные особенности острой внебольничной пневмонии у беременных женщин. *Патология (Ташкент)*, *3*, 48-51.

32. Наврузова, Р. С., Султанов, С. Н., & Юсупова, М. А. РАННЯЯ ДИАГНОСТИКА ХРОНИЧЕСКИХ ЦЕРВИЦИТОВ, АССОЦИИРОВАННЫХ С ВИРУСОМ ПАПИЛЛОМЫ ЧЕЛОВЕКА У БЕРЕМЕННЫХ ЖЕНЩИН. *ООО «Maxliyo-shifo» & V*, *87*.

33. Раджапова, Д. Д., & Авезов, М. И. (2017). Наш опыт лечения полипозного риносинусита. *Научный альманах*, (1-3), 226-229.

34. Рахманова, И. И., & Юсупова, М. А. (2017). ПРОВОСПАЛИТЕЛЬНЫЕ ЦИТОКИНЫ В ПАТОГЕНЕЗЕ ВНЕБОЛЬНИЧНОЙ ПНЕВМОНИИ У БЕРЕМЕННЫХ. *МОЛОДЫЕ УЧЕНЫЕ–МЕДИЦИНЕ*, 211.

35. Юсупова, М. А. (2005). Роль провоспалительных цитокинов интерлей-кина-1р и фактора некроза опухоли а в патогенезе внеболь-ничной пневмонии у беременных. *Иммунология*, *26*(6), 345-345.

36. Юсупова, М. А. (2018). ОПЫТ РОДОРАЗРЕШЕНИЯ БЕРЕМЕННЫХ ЖЕНЩИН С ТЯЖЕЛОЙ ВНЕБОЛЬНИЧНОЙ ПНЕВМОНИЕЙ (ТВП). *САНКТ-ПЕТЕРБУРГСКИЙ СЕПТИЧЕСКИЙ ФОРУМ-2018*, 62.

37. Юсупова, М. А., & Мадаминова, З. Ю. (2019). Эффективность фитоэстрогенов в лечении климактерического синдрома. In *Научные разработки: евразийский регион* (pp. 164-167).

38. Юсупова, М. А., & Махмудова, М. А. (2016). Стилистика фразеологических единиц в английском и русском языках. *Ученые записки Худжандского государственного университета им. академика Б. Гафурова. Гуманитарные науки*, (1 (46)), 101-104.

39. Юсупова, М. А., Бекметова, Ш. К., & Хайтбоев, Ж. А. (2017). Распространенность заболеваний шейки матки у беременных женщин в Узбекистане. *Universum: медицина и фармакология*, (3 (37)), 4-8.

40. Юсупова, М. А., Джуманиязова, Г. М., & Ходжаева, З. К. (2017). Сравнительная оценка методов диагностики болезней шейки матки у беременных. *Research'n Practical Medicine Journal*, (S2), 105.

41. Юсупова, М. А., Исмаилова, Д. У., & Матмуратова, С. О. (2017). Алгоритм ведения беременных с патологией шейки матки. *Интерактивная наука*, (12), 84-88.

42. Юсупова, М. А., Ходжаева, З. К., & Шарафаддинова, Г. Р. (2017). Клинико-рентгенологические показатели при респираторном дистресс-синдроме у беременных с внебольничной пневмонией. *Авиценна*, (5), 4-8.