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REVIEW

The role and significance of the oncobiota in selected cancers: a review

Łucja Justyna Walczak[1] · **Urszula Kosikowska**[2] · **Mariola Herbet**[1]

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Abstract
This review provides an overview of research evidence focused on the microbial components essential to clinical cancer care, called the oncobiota (the interaction of human microbiota and cancer cells). It specifically examines the oncobiota in central nervous system cancer,breast cancer, pancreatic cancer, liver cancer, lung cancer, and cervical cancer. The literature review reveals insufficient knowledge about the oncobiota of organs once considered sterile. Many studies on oncobiota focus on small, geographically specific patient groups, and the absence of a reference (control) group complicates the development of microbial profiles for selected cancers. Consequently, this review aims to analyze the literature data and reports on the role of oncobiota in selected "sterile" organs and the resulting therapeutic or preventive implications. All relevant publications on oncobiota in patients with the selected cancers were considered to provide the most thorough analysis possible. Understanding the significance and role of oncobiota in the pathomechanisms of carcinogenesis may pave the way for targeted cancer prevention methods. Furthermore, therapeutic strategies based on oncobiota could represent a novel area of personalized cancer treatment. Additionally, oncobiota may serve as an additional diagnostic tool in oncology.

Keywords Human microbiota · Oncobiota · Oncobiome · Cancer · Carcinogenesis

Introduction

Cancer is the third leading cause of death worldwide, following cardiovascular and respiratory diseases [1]. The cancer process is highly complex and varies based on the carcinogen, cancer type, and patient-specific factors [2, 3]. Due to this heterogeneity, despite significant medical advances, a selective anti-cancer drug has yet to be discovered. Therefore, it is essential to pursue ongoing research to better understand the cancer process, including the mechanisms involved in carcinogenesis—such as enzymes, proteins, genes, ultraviolet radiation, microbiota, and other contributing factors. This understanding may, in the future, enable the development of drugs targeting specific therapeutic pathways.

Reports on the role of the microbiota (formerly microflora or natural flora) and the microbiome in cancer development are increasing. It is estimated that abnormal microbial composition and function—characterized by microbiota dysbiosis and the presence of specific organisms—may be associated with carcinogenesis in approximately 15–20% of cancers [4, 5].

It is important to clarify the distinction between the microbiome and microbiota. By definition, the microbiota are all microorganisms (primarily bacteria, fungi, and some protists) inhabiting a specific environment, such as an organism, system, or tissue [6]. In contrast, the microbiome encompasses not only the microbiota but also their collective genomes and proteomes [7]. The term "oncobiota" specifically refers to the interaction and composition of human microbiota and cancer cells. However, this term has yet to be universally standardized in the literature [8]. Neoplastic diseases are characterized by abnormal microbiota/microbiome composition and function, contributing to their pathogenic role.

Much of the research has focused on the gut microbiota, which has consistently been shown to play a crucial role in

✉ Łucja Justyna Walczak
lucja.walczak@umlub.pl

✉ Urszula Kosikowska
urszula.kosikowska@umlub.pl

[1] Chair and Department of Toxicology, Faculty of Pharmacy, Medical University of Lublin, 8 Chodźki Street, 20-093 Lublin, Poland

[2] Department of Pharmaceutical Microbiology, Faculty of Pharmacy, Medical University of Lublin, 1 Chodźki Street, 20-093 Lublin, Poland

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the development of many malignancies. The composition of the gut microbiota varies according to several factors, including gender, comorbidities, diet, cancer type, and others [9–12]. Many studies have described distinct patterns. For example, lung cancer patients have been shown to have a lower ratio of *Bacillota* to Bacteroidetes in their gut microbiota compared to the healthy control group [13].

In the same study, the microbiota of the lung cancer groups exhibited a higher abundance of *Pseudomonadota* and *Verrucomicrobia* compared to the control group. In contrast, patients with hepatocellular carcinoma (HCC) displayed an increased abundance of *Bacteroides* spp., *Phascolarctobacterium* spp., *Enterococcus* spp., *Streptococcus* spp., *Gemella* spp., *Bilophila* spp. [14]. Conversely, reduced levels of *Akkermansia* spp., *Bifidobacterium* spp., *Dialister* spp., *Collinsella* spp., and *Adlercreutzia* spp. were found in comparison to the control group. The same study demonstrated that the microbiota profile in HCC patients was closely correlated with inflammation. In a mouse model of glioblastoma, the abundance of the *Verrucomicrobia phylum* and the *Akkermansia genus* in the gut microbiota was shown to increase following glioblastoma growth [15]. Futhermore, several microorganisms closely associated with the process of carcinogenesis have been discovered and characterized.

These discoveries hold significant potential to revolutionize cancer treatment. It has long been established that colonization of the stomach by *Helicobacter pylori* could consequently lead to the development of gastric cancer [16]. Another bacterium with proven carcinogenicity in the liver is *Helicobacter hepaticus* [17]. This microorganism is a risk factor for the progression of liver disease to cirrhosis and hepatocellular carcinoma, particularly in patients chronically infected with the hepatitis C virus. In a mouse study, this bacterium was shown to induce mammary adenocarcinoma [18]. *Citrobacter rodentium* is another microorganism closely associated with carcinogenesis [19]. Infection with this bacterium has been shown to promote colon tumorigenesis in Apc(Min/+) mice, a widely used model of colon cancer [20]. Similarly, *Salmonella enterica* has been observed to promote the transformation of genetically predisposed cells and is a major carcinogen in biliary tract cancer [21]. Notably, approximately 40% of patients with documented *Streptococcus bovis* bacteremia have been found to have colorectal or extra-colonic cancer over the last twelve years [22]. These findings highlight the potential for new treatments and preventive strategies targeting specific microorganisms.

Understanding the qualitative and quantitative composition of the oncobiota, as well as the differences between the oncobiota and microbiota of healthy individuals, is crucial for identifying changes caused by disease or therapeutic interventions [23, 24]. The microbiota performs protective, defensive and immunostimulatory functions. Alterations in the oncobiota compared to the microbiota of a healthy person can significantly impact the efficiency of the patients' immune systems and their susceptibility to various diseases, including infections. Knowledge in this area can facilitate the development of personalized treatment regimens, including the introduction of tailored diets to modulate the oncobiota and improve patients' quality of life. Additional approaches, such as the use of probiotics or fecal therapy, may also play a role in influencing the microorganisms inhabiting the human body. Increasingly, biological markers are being identified through modern diagnostic methods and targeted tests to evaluate patients' health status and monitor therapeutic effects.

Advancements in molecular biological techniques, such as next-generation sequencing (NGS) or targeted sequencing of the 16S ribosomal RNA (16S rRNA) gene, have significantly expanded our understanding of the human microbiome [25–27]. Until recently, organs such as the brain, liver, or ovary were considered sterile regions of the human body. This assumption was based on their perceived lack of microorganisms or the inability to culture microbes from these regions using routine microbiological methods. As a result, microorganisms detected in diagnostic materials obtained from these organs were often dismissed as contaminants [28, 29]. Their presence was attributed to either the aspiration of microorganisms from other parts of the body or irregularities in the collection of diagnostic samples.

The increasing number of deaths from malignant tumors, coupled with the development of new molecular techniques, underscores the urgent need to expand knowledge in this area. Changes in the microbiota of people with malignant tumors are increasingly recognized, with growing emphasis on their role in preventing and treating these conditions [25, 30, 31].

The role of the human microbiota in tumorigenesis is highly complex. However, most data on the role of the microbiota in therapy, including immunotherapy of certain diseases, has been derived from studies of the gut microbiota [24, 32]. For instance, altering the composition of the gut microbiota—through dietary interventions, probiotics, or fecal therapy—has been shown to enhance its immunomodulatory functions and contribute to personalized treatment approaches [23]. One study demonstrated that *Salmonella* spp. can be used as a carrier of the cytosine deaminase gene to malignant tissues, which affects the metabolism of 5-fluorouracil, an anticancer drug [33]. Additionally, gut bacteria are suggested to function as immune checkpoint modulators [34]. Studies in colorectal cancer have shown that the gut microbiota can help mitigate the treatment side effects [35]. Furthermore, microbiota present in the tumor microenvironment may contribute to cancer progression and influence variability in therapy responses among patients [24].

Recent reports indicate that specific bacteria can be found in the microbiota of various human cancers. The

term 'oncobiota' has also been coined [26]. It refers to the study of the interaction between the human microbiota or microbiome and carcinogenesis. 'Oncobiome' is a broader term that refers to the study of the interaction between the human microbiome and carcinogenesis, including genomic studies and standard culture methods. [36]. It is worth noting that these definitions are being supplemented or modified in accordance with the increasing knowledge on the subject, gained with the use of modern research tools [6]. The term encompasses all microorganisms that contribute to the process of tumorigenesis. Furthermore, until recently, the commensal microbiota of neoplastic tissues was thought to occur only in tumors in direct contact with the environment, such as gastrointestinal cancers [37].

Therefore, this review aims to analyze literature and reports on the role of oncobiota in selected "sterile" organs and the resulting therapeutic or preventive implications. The article focuses on oncobiota in patients with central nervous system cancer, breast cancer, pancreatic cancer, liver cancer, lung cancer, and cervical cancer. All publications that addressed oncobiota in patients with selected cancers were considered. These efforts aimed to analyze the topic as comprehensively as possible. There is a lack of papers in the literature comparing oncobiota across different types of cancer; most studies are limited to a kind of cancer, or if they do compare, they often focus on the gut microbiota.

The role of microorganisms in tumours of the central nervous system (CNS)

Gliomas are the most common primary brain tumors in adults, accounting for more than 80% of all central nervous system (CNS) malignancies. [38–40]. These tumors are characterized by high malignancy, significant mortality, and a high risk of recurrence. The World Health Organisation (WHO) classifies them into fifteen entities of diffuse adult gliomas based on molecular and histological diagnosis. Among them, three main types are distinguished: astrocytoma, ependymoma, and oligodendrogliomas. Since the brain is considered, in principle, to be a 'sterile' organ, various theories of the origin of the glioma microbiota have beem emerged. One theory suggests that bacteria may already exist in brain tissues before the tumor occurs, potentially including the initiation and migration of gliomas [37]. The presence of microbiota or microorganisms in the brain tissue of healthy patients and those with various non-infectious CNS-related diseases support this. An example of such a study is one that examined brain sections from people with HIV/AIDS but also those with other brain diseases [41]. Using NGS, bacterial 16S RNA sequences were detected in all samples. Seventy percent of these sequences involved *alpha-proteobacteria* (*Pseudomonadota* being the primary phylum).

In contrast, another study also investigated the composition and abundance of the microbiota/microbiome in frozen and fixed autopsy brain samples from patients with multiple sclerosis (MS) and patients without MS [42]. Both groups showed 1200–1400 bacterial genomes/cm^3 and low bacterial rRNA:rDNA ratios in the brain white matter. Moreover, *Pseudomonadota* specific rRNA sequences predominated in the MS group. Similar findings were observed in the brains of Alzheimer's disease (AD) patients and controls [43]. Lipopolysaccharide (LPS) and *Escherichia coli* K99 pilus protein was shown to be present in both brain parenchyma and vessels, and their levels (LPS and K99) were higher in sections from AD patients. Furthermore,16SNGS-based high-resolution 16S rRNA PCR revealed that bacterial rRNA levels differed in various brain areas associated with AD and Parkinson's disease [44]. The sites of brain involvement and bacterial profiles differed according to disease stage. In contrast, some studies suggest that gliomas may alter the local microenvironment, allowing bacteria to invade the tumor from elsewhere (destruction of the blood–brain barrier (BBB)), or bacteria may gain access to the brain via neuronal transport via the trigeminal nerve, olfactory and facial nerve and the vagus nerve [37]. Another possibility is that bacteria are transported into tumor-containing tissues along with immune and cancer cell migration. The breakthrough study on glioma oncobiota used PCR sequencing of multiplexed 16S bacterial 5R rDNA to achieve species-level resolution [45]. In this study, *Pseudomonadota* were predominantly dominated in glioma tissue, followed by *Bacillota, Acinobacteria, Bacterioides, Fusobacteria, Cyanobacteria*, and others. At the species level, the most abundant species observed in glioma cells were *Enterobacter cloacae, Neisseria macacaeand Acinetobacter* US_424strain. The presence of microbiomes in glioma has also been confirmed in another study [46]. As before, LPS was detected, while lipoteichoic acid (LTA) was absent. In addition, sporadic distribution and irregular shapes of LPS fluorescence signals were shown, mainly located near the nucleus or scattered in the intercellular spaces of glioma cells.

The role of microorganisms in breast cancer

Among women, breast cancer is the most commonly diagnosed cancer [47–49]. It accounts for more than one in ten new cancer diagnoses each year. In 2020 alone, two million new cases were reported [50]. There are two main types:

- Invasive (IBC): no particular type (NST), luminal breast cancer, HER2-enriched breast cancer, triple-negative breast cancer (TNBC), and Claudin-Low Breast Cancer;

- Non-invasive: lobular carcinoma in situ (LCIS) and ductal carcinoma in situ (DCIS).

It has been suggested that the breast tissue microbiota may promote the maintenance of healthy breast tissue by stimulating resident immune cells [51]. Three mechanisms are thought to explain the presence of the microbiota in breast tissue [52]. These include translocation through the oral, intestinal, genitourinary, and skin membranes, entry through the bloodstream via oral hygiene care, and dendritic cell sampling of the intestine and transfer through the bloodstream and mesenteric lymph nodes. It is noteworthy that breast tissue is composed of fatty tissue, is richly vascularised, and has abundant lymphatic drainage, making it a suitable candidate for bacterial growth [53].

Eighty-one women with and without breast cancer were examined for the presence of microbiota [54]. None of these women had symptoms of infection. Routine culture methods even isolated some microorganisms. The diversity of microbial taxa was shown to depend on the origin of the women. Canadian women's samples showed that *Bacillus* spp. (11.4%), *Acinetobacter* spp. (10.0%) and *Enterobacteriaceae* (8.3%) were present (Fig. 1). In contrast, samples from Irish women showed *Enterobacteriaceae* (30.8%), *Staphylococcus* (12.7%), and *Listeria welshimeri* (12.1%) as the most prevalent. *Propionibacterium* (10.1%) and *Pseudomonas* (5.3%) were the less abundant taxa.

In contrast, another study compared the microbiota of healthy breast tissue and breast cancer tissue [51]. As before, it was shown that 96.6% of the total sequences in both groups consisted of five clusters: *Pseudomonadota, Bacillota, Actinobacteria, Bacteroidetes,* and *Verrucomicrobia*. This study revealed that tumor tissue had a lower bacterial load than healthy tissue. Furthermore, the bacterium *Methylobacterium radiotolerans* was observed to be relatively enriched in tumor tissue, while healthy tissue showed relative enrichment in paired normal tissue with the bacterium *Sphingomonas yanoikuyae.*

It is worth noting that similar observations were made in another study [55]. This study investigated dysbiosis in sentinel lymph nodes of breast cancer patients, showing a higher abundance of *Methylobacterium radiotolerans* in these patients. The abundance of this bacterial species in tumor tissue was also confirmed in another study [56].

On the other hand, a study in a population of Chinese women found that the type and abundance of microorganisms varied according to both the type and the degree of malignancy of breast cancer [57]. It was shown that neoplastic tissue with a high degree of malignancy was abundant in the genus *Propionicimonas* and the families *Micrococcaceae, Caulobacteraceae, Rhodobacteraceae, Nocardioidaceae, Methylobacteriaceae* (ethnospecific determinants). It was also noted that the relative

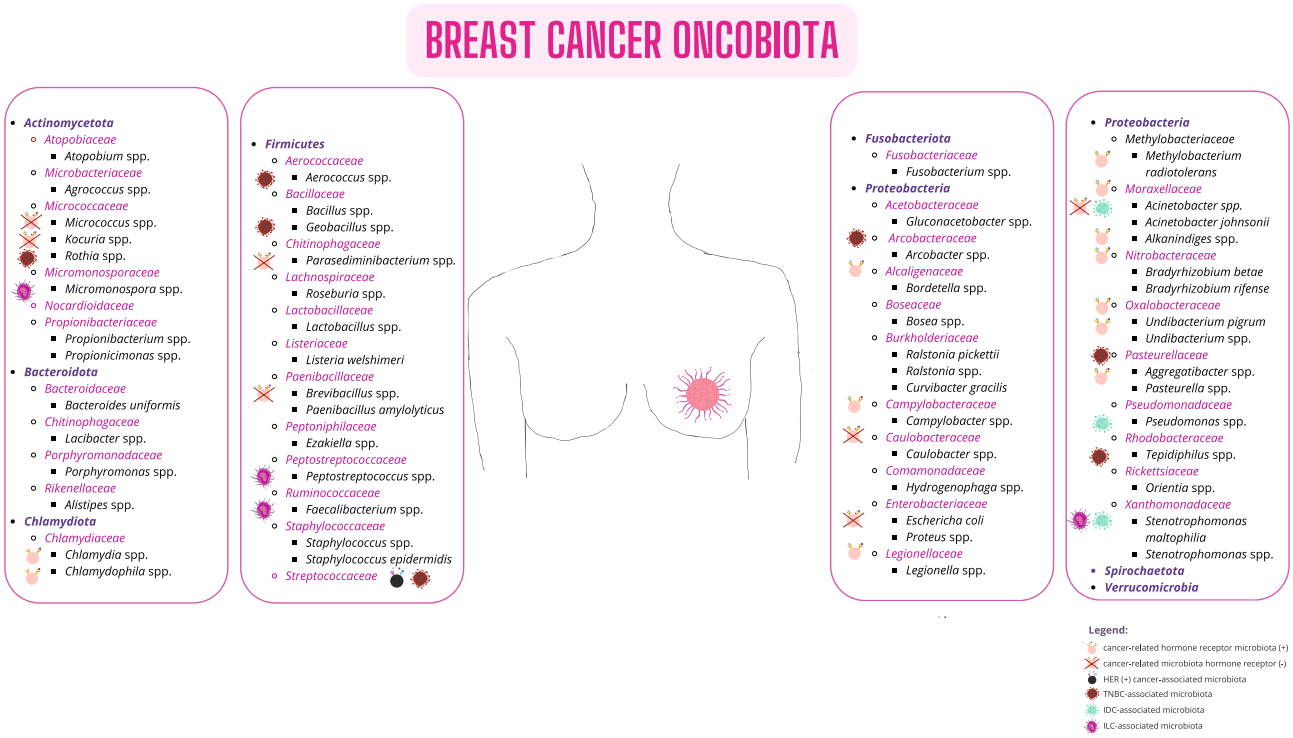

Fig. 1 Breast cancer oncobiota

abundance of the family *Bacteroidaceae* and the genus *Agrococcus* increased with tumor growth.

Furthermore, another report showed that the microbiota of breast tissue changes as the composition of the breast tissue changes (percentage of fat and percentage of fibrosis) [58]. *Bacillota* (at the genus level, *Staphylococcus*) were positively correlated with the fat percentage. A negative association with the *Bacillota* phylum and a positive association with *Spirochaetes* were observed for the rate of fibrosis.

Using 16S rDNA hypervariable tag sequencing, it was shown that the breast tissue microbiome was distinct from the overlying skin microbiota and the buccal microbiota, with greater species richness [59]. Furthermore, it was shown that the microbiota of normal breast tissue adjacent to invasive cancer significantly differed from that of normal breast tissue adjacent to benign disease. Specific taxa at the genus level were significantly enriched breast tissue from women with cancer, including the genera *Fusobacterium, Atopobium, Gluconacterobacter, Hydrogenophaga,* and *Lactobacillus.*

Meanwhile, another study found that women with breast cancer had a higher relative abundance of *Bacillus* spp., *Enterobacteriaceae,* and *Staphylococcus* spp. [60]. It was also observed that lysed *Escherichia coli* and *Staphylococcus epidermidis* could cause DNA damage in vitro. In contrast to the previous study [59], the microbial profiles were similar between normal adjacent tissue and tissue taken directly from the tumor.

According to another investigation involving 221 breast cancer patients, 18 breast cancer predisposed patients (based on genetic predisposition or medical history), and 69 healthy controls, the genera *Anaerococcus, Caulobacter, and Streptococcus*, which were significant bacterial hubs in benign tissue networks, were absent from cancer-associated tissue networks [61]. In contrast, *Staphylococcus* spp. and *Propionibacterium* spp. were observed to be depleted in tumor tissue and negatively associated with oncogenic immune features. However, the genera *Streptococcus* and *Propionibacterium* were positively correlated with genes related to T-cell activation. Notably, in all tissue types, the *Pseudomonadota* group was the most abundant bacterial phylum, followed by *Bacillota* and *Actinobacteria*. Differences were only observed at lower taxonomic levels. Tumor tissue contained a significantly higher proportion of the families *Pseudomonadaceae* and *Enterobacteriaceae*. The abundance and composition of the microbiota also differed according to the level of complexity. For example, the genera *Porphyromonas, Lacibacter, Ezakiella,* and *Fusobacterium* were more abundant in higher and lower-stage cancers. The presence of various species of *Tepidiphilus, Alkanindiges,* and *Stenotrophomonas* characterized invasive ductal carcinoma (IDC). In contrast, invasive lobular carcinoma (ILC) samples contained bacteria from the genera *Peptostreptococcus, Micromonospora, Faecalibacterium,* and *Stenotrophomonas*. It was also observed that estrogen receptor-positive tumors consistently had a lower abundance of seven genera (*Alkanindiges, Micrococcus, Caulobacter, Proteus, Brevibacillus, Kocuria, Parasediminibacterium*) compared to ER-negative tumors [61].

Another study detected nearly 900 bacterial species from four predominant phylum: *Pseudomonadota, Bacillota, Actinobacteria,* and *Bacteroidetes* [62]. At the family level, bacteria from *Burkholderiaceae* dominate, followed by *Sphingomonadaceae* and *Alcaligenacea. Ralstonia pickettii* was the most abundant species in all breast tissues, and its relative abundance increased with decreasing malignancy. Notably, the abundance of *Curvibacter gracilis* was lower in breast cancer tissue without hormone receptors. In contrast, the order *Pseudomonadales, Bradyrhizobium betae, Acinetobacter johnsonii, Bradyrhizobium rifense, genus Acinetobacter,* family *Moraxellaceae, Undibacterium pigrum,* and genus *Undibacterium* were predominant in breast cancer tissues with hormone receptors.

In addition, a higher abundance of the genus *Ralstonia* was also observed in non-Hispanic black women [63]. As in other studies, the *Pseudomonadota* phylum had the highest abundance, followed by *Bacillota, Bacteroidetes,* and *Actinobacteria* in all tissue types (tumor, regular, and adjacent tissue). The TNBC tumor showed an enrichment of the *Streptococcaceae* family. Furthermore, the higher abundance of the genus *Bosea* increased with the stage of breast cancer.

In contrast, another study showed that *Ralstonia* was the predominant genus with no significant differences in abundance between tumor and normal tissue [64]. In addition, tumor tissue was found to have a significantly lower microbial diversity than healthy tissue, consistent with previous reports [51]. This study also confirmed that *Pseudomonadota* were the predominant bacteria in all tissues, followed by *Actinobacteria, Bacillota,* and *Bacteroidetes.* This study also showed racial differences in the tissue and breast cancer microbiota.

Furthermore, another study confirmed that the *Pseudomonadota* and *Actinobacteria* phylum were predominant in cancerous and healthy breast tissue [65].

Propionibacterium and the *Propionibacterium* enrichment culture clone MRHull-FeSM-11R and *Propionibacterium acnes* were more abundant in non-tumor tissue. *Bacillota* and *alpha-proteobacteria* were found in higher values in tumor tissues. It was also confirmed that tumor tissue showed a lower microbial diversity than healthy tissue. In both tissue types, *Propionibacteriaceae*, *Moraxellaceae*, and *Pseudomonadaceae* were observed to be the most abundant at the family level. In particular, one study showed that different breast cancer subtypes are characterized by specific oncobiota [66]. Specific taxa were also shown to correlate with patient survival and prognosis. The microbiota of

TNBC was the least differentiated from the other tumors and was represented by *Aggregatibacter spp,* whereas HER + samples showed the most abundant oncobiota with more bacterial signatures (mainly *Pseudomonadota*).

The researchers suggest that these findings may be necessary to develop future treatment strategies targeting the microbiota. A correlation between microbiological signature and breast cancer type was also found in another study [67]. In triple-positive breast cancer tissue, significantly higher signals were found for the bacterial genera *Bordetella, Campylobacter, Chlamydia, Chlamydophila, Legionella*, and *Pasteurella*. In contrast, for TNBC, the genera *Aerococcus, Arcobacter, Geobacillus, Orientia,* and *Rothia* were significantly associated with this tumor type. At the same time, *Streptococcus* spp. was associated with HER + tumors. It is worth noting that using leave-one-out cross-validation of the predictive model, the microbiological signature was found to identify malignant breast cancer tissue with an accuracy of approximately 85% [68].

A theoretical study used the Sloan neutral community model to define neutral and non-neutral species in healthy (control), benign, and malignant tumor cohorts and explain the dynamics of neutral species [69]. The researchers suggest that stochastic drift and deterministic selection are essential in shaping the structure and dynamics of bacteria in breast tissue, including influencing tumor development.

Nipple aspiration fluid from women with breast cancer and healthy women (control group) was also investigated for microbiota [70]. The genus *Alistipes* was found to be predominant in women with breast cancer. In contrast, bacteria from the *Sphingomonadaceae* family predominated in healthy women. Swabs were also taken from the skin around the nipple, and no differences were observed between the microbiomes of the two groups. In the case of breast cancer patients, microbes that produce beta-glucuronidase (carcinogenic enzyme in gastrointestinal cancers), such as *Roseburia* spp., *Bacteroides uniformis, Paenibacillus amylolyticus,* and the *Rikenellaceae* family and order *Solirubrobacterales* were more common.

The role of microorganisms in pancreatic cancer

Pancreatic cancer refers to a cancer that starts in the pancreatic duct cells [71–73]. It is predicted that by 2030, pancreatic cancer will be the second most common cause of cancer-related deaths [74]. It is worth noting that it was once thought that because of the highly alkaline nature of pancreatic juice and the presence of proteases in the pancreas, no microorganisms could survive [75].

The pancreatic duct directly connects the pancreas to the gastrointestinal tract [76, 77]. The pancreatic intra-cancer microbiome/microbiota may originate from the gut microbiota via the circulatory system or the biliary/pancreatic duct, and the gastrointestinal microbiota is susceptible to environmental, dietary, and lifestyle factors. Most microorganisms associated with the pancreatic tumor microbiome overlapped with the gut microbiota, suggesting bacterial translocation from the gut to the pancreas [78].

A research study has shown that the microbiota of pancreatic ductal adenocarcinoma is characteristic of the stage at which the tumor has progressed [79]. Thirteen different types of microorganisms have been observed in the most significant number of tumors in the human body. Regarding genera*, Pseudomonas,* and *Elizabethkingia* bacteria were the most abundant (Fig. 2.). The same study showed that the microbiota might play a role in disease progression by inducing immune suppression within the tumor. Furthermore, modifying and targeting the pancreatic cancer microbiota contributed to increased susceptibility to immunotherapies and protection against tumorigenesis. This mouse study also showed that gut bacteria can invade pancreatic tissue.

It has also been shown that most bacterial species in pancreatic tumors are intracellular [80]. Pancreatic tumor tissue predominantly harbors microbes associated with oral or gastrointestinal pathology. The most common bacteria found in cancer samples are *Campylobacter* spp. These include *Fusobacterium nucleatum, Leptotrichia* spp.*,* and *Clostridioides difficile.* Furthermore, only one in twenty-seven pancreatic tissue samples tested positive for bacteria. It was also observed that the immune cells had non-specific interactions with the bacteria in the tumor. The researchers suggest that a subset of pancreatic tumors have an altered microbiota with possible bacterial tropism towards malignant tumor cells.

The 16S rRNA gene sequencing was performed on the pancreatic tumor tissue and normal adjacent tissue [74]. Bacteria from the following classes were more common in tumor tissue: *gamma-proteobacteria* (31.2%), *Bacteroides* (14.1%), *Clostridia* (12.8%), *Bacilli* (12.2%), and *alpha-proteobacteria* (11.7%). The composition of the tumor microbiota was found to vary between individuals, as well as the associated risk factors and tumor stage. In never-smokers and those with intermediate cancer stages, the predominant genera were: *Herbaspirillum, Brucella, Allorhizobium, Neorhizobium, Pararhizobium, Rhizobium,* belonging to the *Pseudomonadota* phylum, while the genera *Treponema (Spirochaetota* phylum*), Pseudorhodoplanes,* and *Sphingomnas (Pseudomonadota* phylum*)* were expressed at higher levels in early-stage patients and healthy participants. At the phylum level, the intratumoral and oral microbiota of cancer patients were dominated by *Bacillota, Pseudomonadota,* and *Bacteroidetes.* A higher percentage of the phylum *Pseudomonadota* (44.3%) was also observed in tumor tissue than in the oral cavity. Furthermore, this study demonstrated the potential migration of bacteria between the oral cavity and

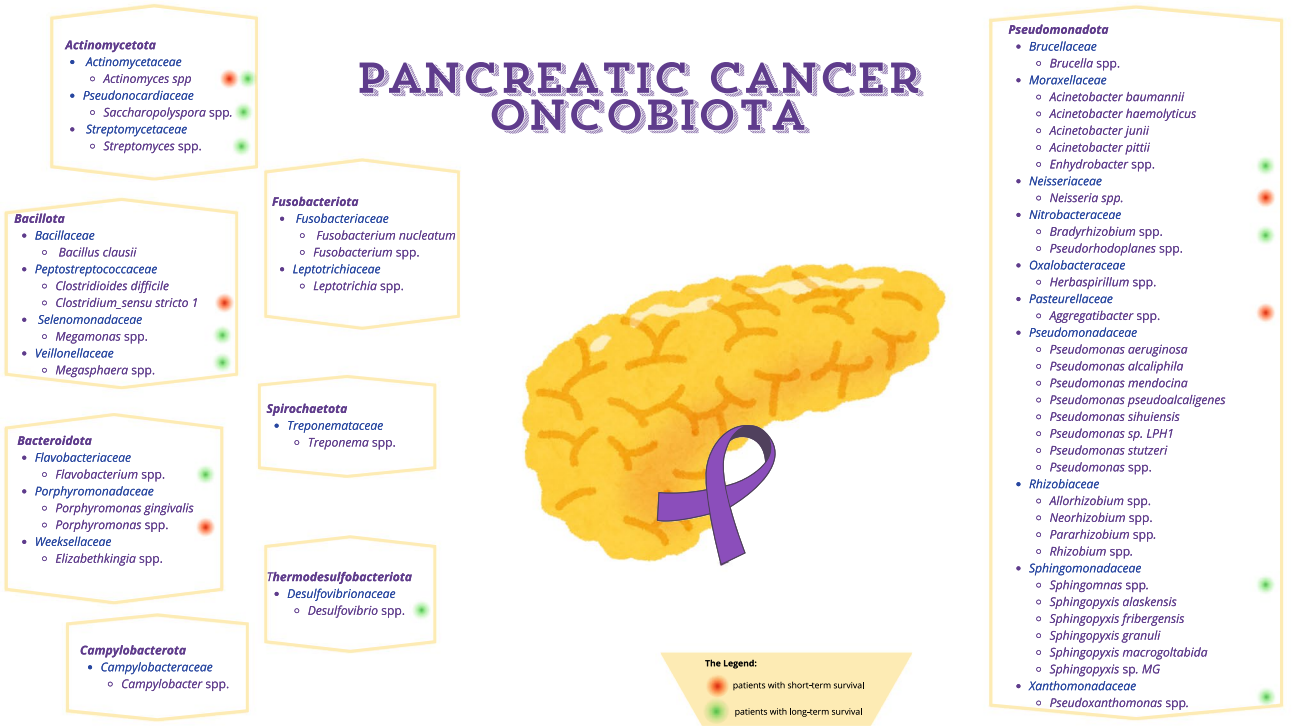

Fig. 2 Pancreatic cancer oncobiota

tumor tissue [81]. In addition, *Porphyromonas gingivalis* was found to promote tumorigenesis.

Another group of researchers analyzed the composition of the pancreatic cancer microbiota in patients with long-term survival (LTS) and short-term survival (STS) [82]. At the genus level, patients with LTS were dominated by species: *Sphingomonas, Megasphaera, Bradyrhizobium, Desulfovibrio, Flavobacterium, Enhydrobacter,* and *Megamonas.* At the same time, samples with STS contained the *Clostridium* sensu stricto 1, *Actinomyces* spp., *Porphyromonas* spp., *Aggregatibacter* spp., and *Neisseria* spp.

Similarly, Riquelme et al. [83] compared the microbiota inside tumors in patients with LTS and STS and found enrichment for specific bacterial communities at different taxonomic levels in each group. At the class level, *alphaproteobacteria, Sphingobacteria,* and *Flavobacteria* predominated in patients with LTS, and *Clostridia* and *Bacteroidea* in those with STS. However, at the genus level, there was no dominant genus in patients with STS, in contrast to patients with LTS, where there was a dominant phylum: *Pseudomonadota (Pseudoxanthomonas)* and *Actinobacteria (Saccharopolyspora* spp. and *Streptomyces* spp.*).* The same study showed that independent of therapy, the diversity and composition of the tumor microbiome/microbiota can influence immune infiltration and, consequently, patient survival. It was also found that *Bacillus clausii*, in particular, can influence patient survival.

In addition, another study showed that *Fusobacterium nucleatum* was the dominant species. Within the species, *Pseudomonadota* dominated [45]. *Fusobacterium* species were found in 8.8% of the tumor tissues examined [84]. Significantly, the researchers found no association between the presence of these microorganisms and any clinical or molecular characteristics. However, the status of *Fusobacterium* spp. in the tumor was independently associated with a worse prognosis during pancreatic cancer. It has also been suggested that these mycobacteria may be a prognostic biomarker in pancreatic cancer [84].

Another study found that the genera *Acinetobacter, Pseudomonas,* and *Sphingopyxis* were highly associated with carcinogenesis [85]. The predominant species were *Acinetobacter* genus: *Acinetobacter pittii, Acinetobacter junii, Acinetobacter baumannii* and *Acinetobacter haemolyticus; Pseudomonas* genus*: Pseudomonas sihuiensis, Pseudomonas stutzeri, Pseudomonas alcaliphila, Pseudomonas pseudoalcaligenes, Pseudomonas* sp. LPH1*, Pseudomonas mendocina, Pseudomonas aeruginosa;* and *Sphingopyxis* genus*: Sphingopyxis macrogoltabida, Sphingopyxis fribergensis, Sphingopyxis granuli, Sphingopyxis* sp. MG*, Sphingopyxis alaskensis.* Increased levels of these species correlated positively with *KRAS* signaling, DNA replication, and pancreatic cancer-related pathways.

Also relevant to treating pancreatic cancer patients is that pancreatic tumor-associated bacteria have been shown

to contain bacteria that can potentially modulate tumor sensitivity to gemcitabine [86]. Therefore, *gamma-proteobacteria* have been reported in pancreatic tissue. Another study showed that adding antibiotics to gemcitabine improved treatment efficacy [87].

The role of microorganisms in liver cancer

Hepatocellular carcinoma (HCC) is one of the most common primary liver cancers (90%) [88–90]. It is the fifth most common cancer in men and the seventh in women. HCC is characterized by a high degree of malignancy and a poor prognosis for patients (five-year survival rate—18%). This type of cancer occurs in 85% of patients diagnosed with cirrhosis. It is important to note that this cancer is usually diagnosed at an advanced stage, which significantly reduces the patient's chances of recovery. Although the liver is considered to be a sterile organ, microorganisms present in the intestine can reportedly enter the liver/intestinal tissues via the portal vein [91].

The microbiota of different subtypes of primary liver cancer was analyzed [92]. LTS tumors showed a predominance of *Pseudomonas* spp., *Thermomonas* spp., and the *Paraprevotellaceae* family (Fig. 3). At the same time, STS tumors had a higher proportion of *Enhydrobater* spp., *Lachnospiraceae,* and *delta-proteobacteria*. Furthermore, it was shown that tumors from patients with HCC and intrahepatic biliary tract cancer were characterized by lower bacterial diversity. It was also shown that the relative abundance of *Enterobacteriaceae* was not significantly correlated with the prognosis of patients with primary liver cancer at the family and genus levels. In contrast, the relative abundance of *Pseudomonadaceae* in the family and genus levels decreased in tumor tissue and was linearly associated with patient survival prognosis.

On the other hand, another study showed that increased microbial counts correlate with poor prognosis [93]. The microorganisms present inside the tumor tissue in hepatitis B (HBV)–positive drinkers have also been shown to be associated with tumor pathway activity. Among these, the researchers list *Caulobacter vibrioides* CB15 and *Herbaspirillum huttiense* subsp. Puter IAM 15032 was associated with the up-regulation of YAP1, TBK1, and the PRC2/EZH2 complexes, while *Anaerococcus prebiotic* DSM 20548 was associated with the up-regulation of the BMI/PRC1 signaling pathway. In contrast, up-regulation of the ATF2 pathway, AKT, and PIGFi down-regulation of P53 was found to be associated with *Staphylococcus epidermidis, Acinetobacter baumanni, Methylorubrum populi* and *Acinetobacter calcoaceticus* in HBV-positive non-drinkers. They were also shown to be associated with a worse prognosis for patients (in addition to *Acinetobacter baumannii*). Xue Ce et al. [94] compared the microbiota of HCC-positive and healthy tissue. Tumour tissue showed a higher percentage of the genus *Acinetobacter*, *Brevundimonas*, *Corynebacterium*,

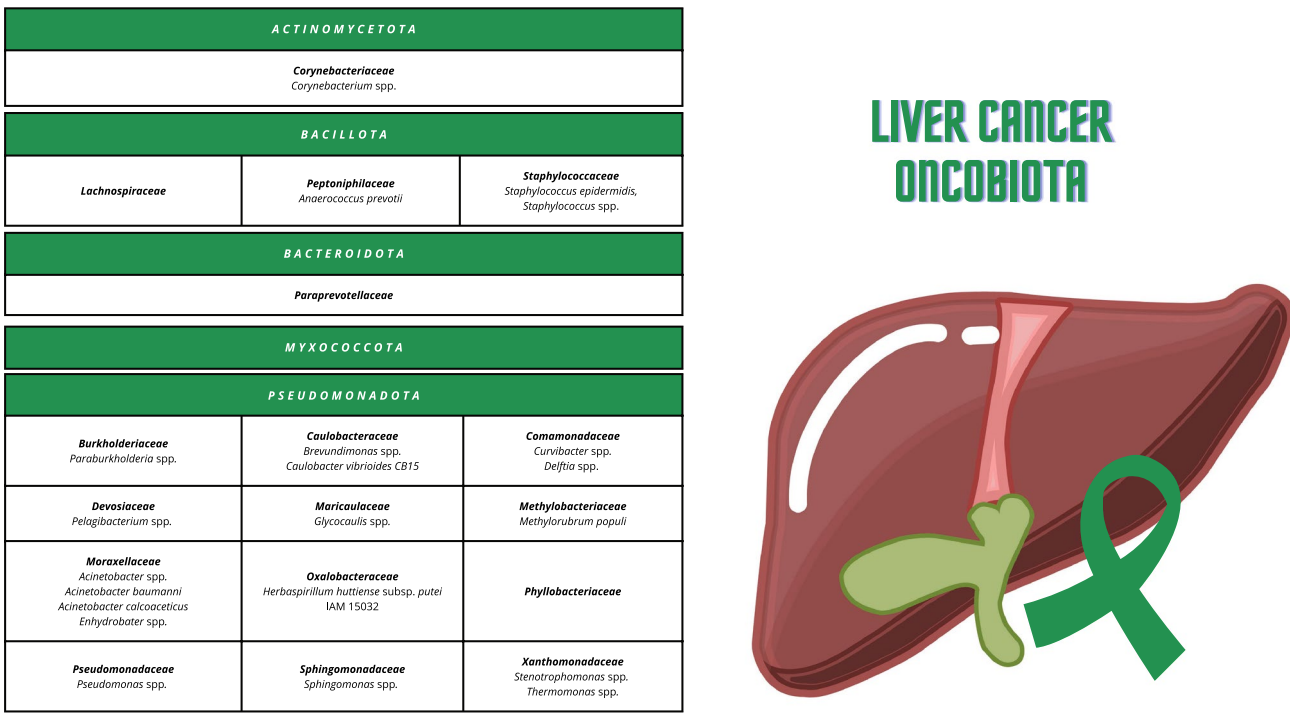

Fig. 3 Liver cancer oncobiota

Curvibacter, *Delftia*, *Glycocaulis*, *Paraburkholderia*, *Pelagibacterium*, *Pseudomonas*, *Sediminibacterium*, *Sphinhomonas*, *Staphylococcus*, *Stenotrophomonas* and at the family level: *Phyllobacteriaceae* and reduced amounts of *Halomonas*, *Nesterenkonia*, compared to healthy tissue.

The role of microorganisms in lung cancer

Lung cancer is one of the most commonly diagnosed cancers, accounting for approximately 12.4% of all cancers [95, 96]. It is characterized by a high rate of metastasis and invasiveness. It is estimated that 90% of lung cancer cases are associated with cigarette smoking. Lung cancer is a heterogeneous disease with a wide range of clinicopathological features. There are two main types: non-small cell lung cancer (NSCLC) and small cell lung cancer (SCLC).

The lung was thought to be a sterile organ. However, studies have shown that the lower respiratory tract microbiota plays a role in developing the immune system in neonates [97, 98]. During the first few weeks of life, *Staphylococcus* spp. or *Ureaplasma* spp. predominate, and as they mature, their microbiota is enriched with other species, such as *Streptococcus, Prevotella, Porphyromonas,* and *Veillonella.* However, some researchers believe most bacterial DNA detected in the lungs may be derived from non-viable bacteria [99].

It is noteworthy that *Bacillota*, *Bacteriodetes*, *Pseudomonadota*, *Fusobacteria*, and *Actinobacteria* are most abundant in the lower respiratory tract of healthy individuals at the family level in the highest numbers, and *Prevotella, Veillonella,* and *Streptococcus* at the genus level [100, 101]. However, the lung microbiota is very diverse and abundant.

It has also been suggested that bronchoalveolar lavage fluid (BAL) samples may well reflect the microbiota of lung cancer [102]. One study found that the genera *Veillonella, Megasphaera, Actinomyces,* and *Arthrobacter* were more abundant in patients with lung adenocarcinoma. In contrast, patients with squamous cell carcinoma were dominated by the genera *Veillonella* and *Rothia* (Fig. 4.). Another study that examined BAL obtained slightly different results and showed that the microbiome of lung cancer patients resembled that of patients with benign lung disease (enrichment in genera: *Capnocytophaga, Sediminibacterium, Gemmiger, Blautia,* and *Oscillospira*) [103].

In contrast, the composition of the lower respiratory tract transcriptome in lung cancer patients was significantly different from that of the control group (enrichment in *Streptococcus* spp. and *Veillonella spp.*), including increased expression of the extracellular signal-regulated kinase (ERK) and phosphoinositide 3-kinase (PI3K) signaling pathways [104]. In another study, BAL analysis showed that the genus *Veillonella* and *Megasphaerawere* enriched in lung cancer patient samples [105].

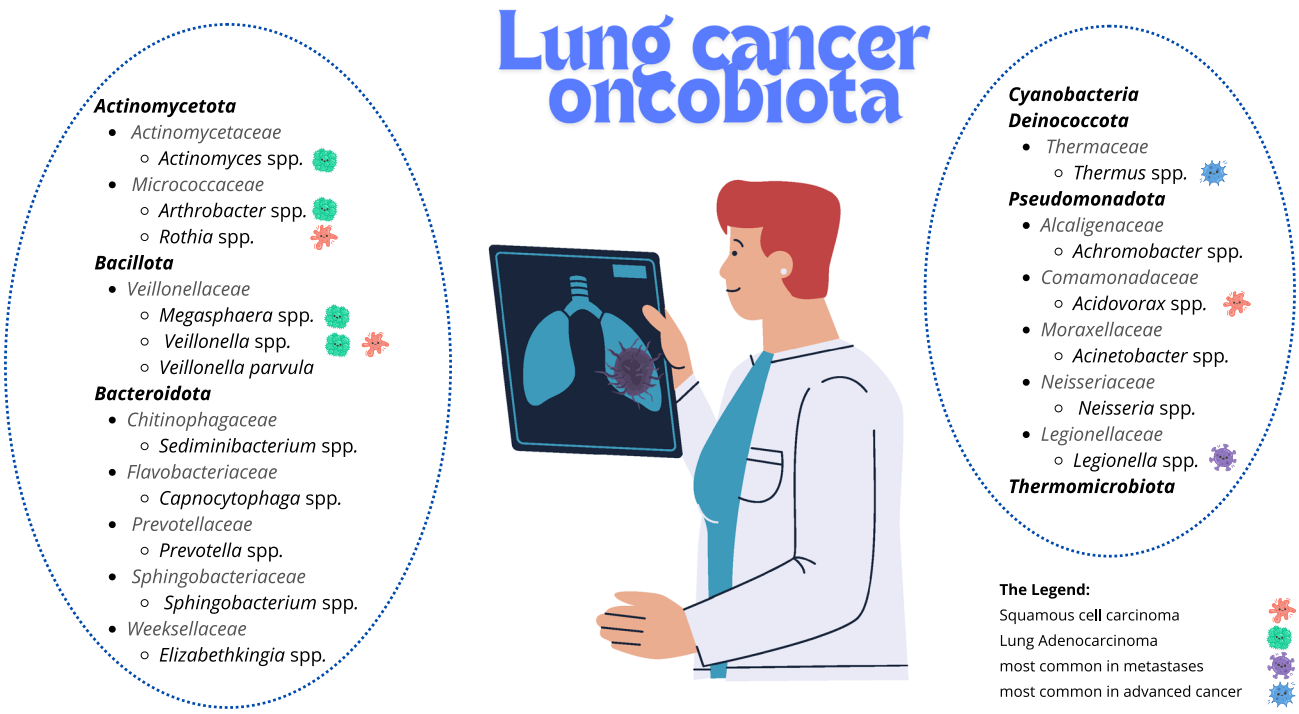

Fig. 4 Lung cancer oncobiota

In another study, the genus S*treptococcus* was also most abundant in lung cancer patients [106]. The genus *Neisseria* was also enriched in tumor lesions, while *Staphylococcus* spp. and *Dialister* spp. were abundant. It decreased progressively from a healthy to a non-cancerous to a cancerous site. In another study, the bacterium *Acidovorax* spp. was shown to be present in the microbiota of the lower respiratory tract of smokers [107]. It was also present in higher numbers in people with squamous cell carcinoma of the lung and TP53 mutations.

Patnaik et al. [108] analyzed BAL, saliva, and resected stage I NSCLC tumor and adjacent lung tissue. The saliva microbiota was characterized by a high abundance of bacteria from the *Bacteroides* and *Clostridia* classes and a low abundance of *alpha-proteobacteria*, *beta-proteobacteria*, and *gamma-proteobacteria.* The latter three classes were most abundant in tissues (cancerous and non-cancerous). In contrast, the BAL microbiota was quite diverse. Some samples contained microbiota similar to saliva and others to tissue (probably due to contamination of BAL with saliva). In the case of tumor tissue, increased *Staphylococcus* spp. and decreased *Bacillus* spp. and *Anaerobacillus* spp. were observed in patients with relapse. On the other hand, when it came to saliva samples, patients with relapses had a higher abundance of the genus *Delftia* and a lower abundance of the genus *Bifidobacterium*. The abundance of the genera *Sphingomonas*, *Psychromonas*, and *Serratia* increased in the relapse group, while the abundance of *Cloacibacterium, Geobacillusand,* and *Brevibacterium* decreased most in the BAL samples. Another study found a different result [109]. It was shown that the microbiota of lung cancer tissue did not affect patient survival. However, the composition and abundance of lung tissue were associated with patient survival and risk of recurrence. It was observed to be less diverse than the paired microbiome of normal tissue but similar in composition to it. Notably, in normal tissue, a higher abundance of the *Lachnospiraceae* family and the *Faecalibacterium* and *Ruminococcus* genera*, Roseburia,* and *Ruminococcus* were associated with reduced recurrence-free and disease-free survival. In contrast, a higher abundance of *Coribacteracea* was associated with increased recurrence-free and disease-free survival. Lung cancer samples were characterized by a higher abundance of the family V*eillonellaceae*, a lower abundance of the genus *Cloacibacterium*, and the family *Erysipelotrichaceae* compared tostandard paired samples.

Veillonella parvula was identified as a species associated with survival in lung cancer patients [110]. Its presence was most abundant and was related to the upregulation of pathways such as IL17, PI3K, MAPK, and ERK, which was consequently associated with tumor progression. Stage IIIB-IV NSCLC patients were also shown to have more significant enrichment of the lower respiratory tract microbiota with oral commensals than stage I-IIIA patients (due to altered immune tone in stage I-IIIA). The role of the commensal microbiota in tumor development and progression was also highlighted in another study [111]. The local microbiota of lung adenocarcinoma has been shown to induce inflammation, stimulate the production of the cytokines IL-1β and IL-23, and consequently contribute to the activation of T γδlymphocytes residing in the lung. It is worth noting that this study was not conducted on humans, but on mice.

Achromobacter, *Acinetobacter*, *Actinomyces*, *Elizabethkingia*, *Rothia*, and *Sphingobacterium* have also been identified in patients with lung cancer cancer [112]. Furthermore, an increased number of cyanobacteria was detected in the same study in these patients. In another study, the phylum *Pseudomonadota* was identified as predominant, together with *Thermi* and *Cyanobacteria*. In addition, the genus *Thermus* was more common in patients with advanced cancer and the genus *Legionella* in patients with metastases [113]. On the other hand, different results were obtained because the lung cancer microbiota was characterised by a lower abundance of the *Pseudomonadota* phylum (mainly the genera *Acinetobacter* and *Acidovorax*) and a higher frequency of *Bacillota* (*Streptococcus* spp.) and Bacteroidetes (*Prevotella* spp.) [114].

The role of microorganisms in ovarian cancer

Ovarian cancer is one of the leading causes of death among women diagnosed with gynecological cancers [115, 116]. It represents the fifth most common cause of death in women. Among others, there are four most common histological types of epithelial ovarian cancer: serous, endometrioid, clear cell, and mucinous tumor.

Using the PathoChip technique in combination with NGS, it was shown that the bacterial signatures in tumor tissue were radically altered compared to matched and unmatched controls, and significantly more bacteria were detected [117]. The tumor tissue detected two predominant bacterial clusters (Fig. 5). These were *Pseudomonadota* (52%), followed by *Bacillota* (22%). In these samples with the highest hybridization signal, the genus *Pediococcus* was present, followed by the genera *Burkholderia, Sphingomonas, Chryseobacterium, Enterococcus, Staphylococcus, Treponema,* and *Francisella.* Notably, the genus *Shewanella* was detected with the highest frequency in 91% of the tumors.

As in the previous study, *Pseudomonadota* is the most abundant phylum, but *Pseudomonas* was the most abundant genus [118]. Furthermore, *Pseudomonas* B14-6, *Klebsiella michiganensis*, *Buchnera aphidicola, Paraburkholderia edwinii, Comamonas aquatica, Veillonella Nakazawa, Corynebacterium jeikeium,* and *Bifidobacterium*

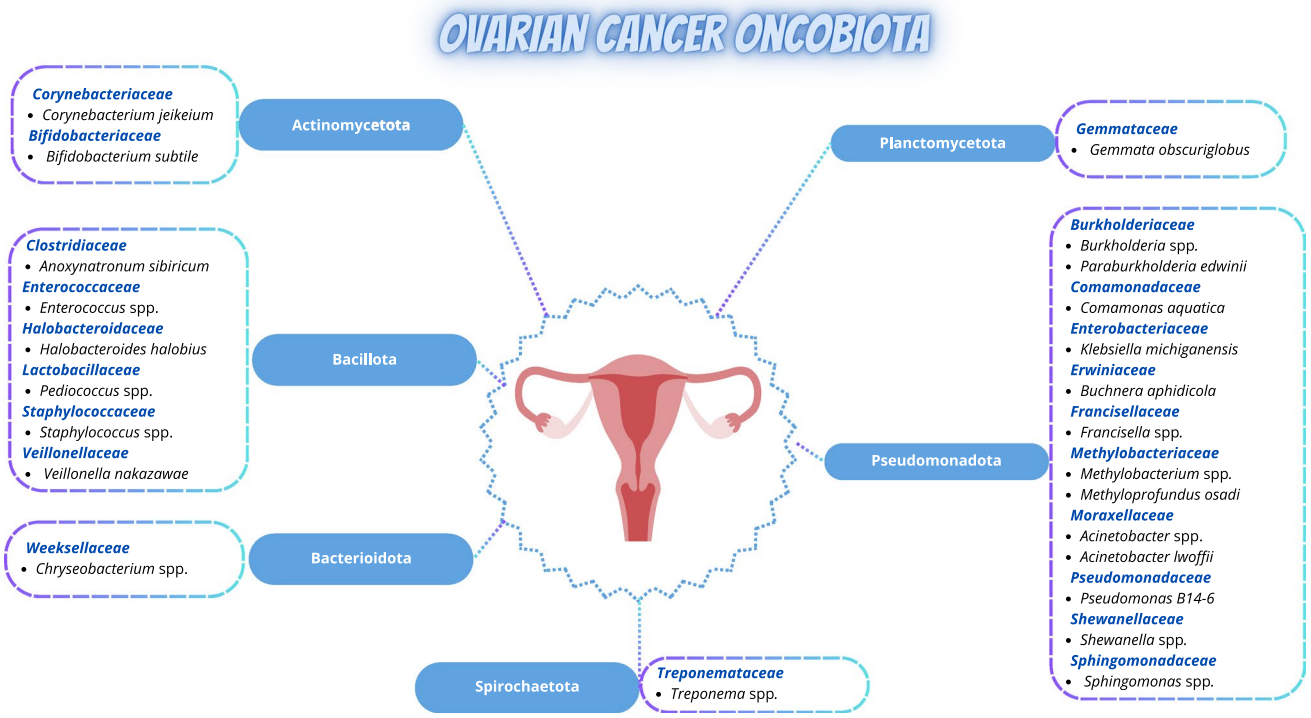

Fig. 5 Ovarian cancer oncobiota

subtile were associated with a worse prognosis, whereas *Gemmatirosa kalamazoonesis* improved the prognosis of these patients This study used the Kraken2 pipeline for microbiome data analysis from RNA sequencing and suggested further studies using metagenome sequencing and/or PCR for more robust findings.

Additionally, Zhou et al. [119] observed that at the cluster level, both tumor tissue and normal tissue taken from the distal part of the fallopian tube were dominated by *Pseudomonadota* and *Bacillota*. Regarding the genus, the dominant taxa in the tumor tissue were *Acinetobacter, Sphingomonas,* and *Methylobacterium,* while *Lactococcus* was predominant in the normal tissue. *Acinetobacter lwoffii* was significantly enriched in ovarian cancer and *Lactococcus piscium* normal tissue at the species level.

Another study found the most common types in both tissues were *Pseudomonadota* (67.1% in the control group and 67.20% in the tumor group), *Bacillota* (23.77% in the control group and 23.82% in the tumor group), and Bacteroidetes (3.26% in the control group and 3.41% in the tumor group) [120]. In normal tissue, *Halobacteroides habits* (14.53%), *Gemmata obscuriglobus* (11.07%), and *Methyloprofundus* (10.69%) dominated at the species level. In contrast, *Gemmata obscuriglobus* (13.89%), *Halobacteroides habits* (11.99%), and *Methyloprofundus osadi* (11.12%) were most abundant in tumor tissue. The same study also showed that *Anoxynatronum sibiricum* may be associated with the cancer stage.

Limitation

This review reveals a lack of sufficient knowledge about the oncobiota of organs once considered sterile.

The number of studies on oncobiota in CNS tumors is limited. To our knowledge, this review includes all studies published to date in this area. Expanding this line of research in the future would be advisable, given the significant findings in this field and the development of new research techniques to create a representative group of bacteria associated with specific types of cancer. In the case of breast cancer oncobiota, only one of the studies used standardized culture methods [54]. This may have affected the diversity of taxa detected. Some microorganisms that are not cultured according to standard procedures may go undetected. Other study did not examine tumor tissue, only nipple aspiration fluid; therefore, it should be noted that the tumor microbiota may not be the same [70]. Noteworthy, racial differences in breast cancer oncobiota. In the presented studies, the microbiota of cancer tissue differed. Chinese, non-Hispanic, Canadian, and Irish female populations were considered. In addition, this also creates some limitations. All the referenced

studies lacked a mixed group consisting of different female populations. In addition, only a few studies have tested the correlation between the type of breast malignancy and the oncobiota.

Research on the microbiome of pancreatic cancer also has several limitations. These include the lack of a counter group and the small size of the study groups. Future studies should identify correlations between relevant bacterial signatures and the type of pancreatic cancer and its stage. In particular, more microbiological studies should be conducted in the early stages of the disease due to the definition of specific bacterial clusters that can accelerate diagnosis.

As in the pancreatic cancer study, the microbiome of liver tissue in healthy subjects was not assessed. Another limitation of one survey of liver oncobiota is that the researchers could not distinguish between live bacteria and nucleic acid fragments derived from them from other body parts [93]. They also used direct alignment instead of 16 s rRNA sequencing, which may not have provided adequate resolution. Moreover, the number of studies on HCC and its oncobiota is severely limited. Two studies of the three cited involve the Chinese population. Only one study was conducted in the United States. Another problem was often the small size of the study sample.

In studies related to lung cancer, few focus on tumor tissue itself. Most studies focus on BAL or sputum analysis, which may not provide a complete picture of the oncobiota in lung cancer. For this reason, some of the samples may have been contaminated on collection. Some of the studies conducted were not conducted on healthy people, and nothing was known about their antibiotic treatment, which could also have affected the results. Furthermore, limited research focuses on the microbiota of specific lung cancer subtypes. Additionally, many studies suffer from small patient sample sizes, which can limit the generalizability of the findings.

In the case of ovarian cancer, the research presented here has several limitations. For ethical reasons, collecting tissues from completely healthy patients was impossible. Additionally, only one study involved patient populations from the United States, while the others focused on patients from China. This geographical limitation highlights the insufficient diversity in the study populations, which may impact the generalizability of the findings.

To summarize, many of these studies in this review evaluated specific patient populations from specific geographic regions, which may have influenced the composition of oncobiota. Moreover, the absence of a healthy control group in many studies makes it difficult to establish baseline microbial signatures for comparison. A standardized microbial map for different cancers is essential to understand the microbiota's role in carcinogenesis better.

Another significant issue is the small sample size in many studies, making it challenging to draw firm conclusions, especially when separating groups based on cancer stages is difficult. It is also important to note that most of these studies utilized NGS which, while powerful, has limitations. NGS alone does not provide insight into the mechanisms through which bacteria may influence carcinogenesis. Future studies that address these limitations, with more extensive and more diverse study populations, and include appropriate control groups and better methodological approaches are essential. Such research would significantly improve the clinical value of these studies and our understanding of the role of microbiota in cancer development.

Summary

There is increasing scientific evidence supporting the significant role of opportunistic microorganisms in the development, progression, diagnosis, prognosis, treatment, care, and prevention of various non-communicable diseases, including cancer. Despite these findings, our understanding of how these microorganisms contribute to carcinogenesis, as well as the variation in human microbiome composition and function, remains limited.

Large human population studies, including those of cancer patients, are urgently needed, focusing on the impact of the microbiota/microbiome in various types of cancer. The role of the oncobiota of cancer patients is far from being understood. It is increasingly recognized that opportunistic microorganisms are major factors influencing the health of patients and the course and outcome of a cancer diagnosis and treatment. Many questions still linger regarding the prevalence, specificity, and stability of oncobiota in treatment or on medication. As research methods and analytic models continue to evolve, the understanding of the human body's microorganisms and environmental factors surrounding us will continue to deepen. Knowledge in the differentiation and modulation of microbiota/microbiome will help explain the role of opportunistic microbes in cancer, which would also validate or refute their causality or complicity in carcinogenesis.

Besides traditional diagnosis, newer solutions have risen to study the oncobiota in search of biological diagnostic markers capable of monitoring patient health, monitoring changes due to diseases, or treatment. Techniques like 16S rRNA gene sequencing and protein profiling prove increasingly crucial for the detection, identification, and profiling of communities of oncobiota.

New techniques, such as amplification, sequencing, bioinformatic analysis, and experimental methods in DNA and RNA-based relative and quantitative oncobiota profiling will be crucially involved in successful oncobiota research. Oncobiota-based therapeutic strategies may open new avenues for personalized cancer treatment. Moreover, the oncobiota

could serve as a valuable diagnostic tool for distinguishing between LTS and STS.

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Data availability No datasets were generated or analysed during the current study.

Declarations

Conflict of interest The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper. The authors declare no competing interests.

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