Original Article

The *new* sexual politics of cancer: Oncoviruses, disease prevention, and sexual health promotion

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Abstract In recent decades, scientists have established a causal link between a number of viruses and a variety of cancers accounting for an estimated 12 to 20 per cent of all cancer cases worldwide. When including all cancers resulting from weakened immune systems due to HIV/AIDS, the number of cancer cases directly or indirectly attributable to viruses rises further. In this article, we examine six cases of virus–cancer connections, selecting those viruses that are also sexually transmitted. These include the well-known case of human papillomavirus (HPV) as well as Hepatitis B and C, the Epstein–Barr Virus, HTLV-1, and KSHV/HHV-8. We examine these viral cancer connections as entanglements among sex, science and biomedicine, specifically exploring the varied places, processes and attributions that infuse this health domain with sexual meanings or banish these from view. We argue that such processes and attributions appear in the shadow cast by the HIV/AIDS epidemic. While virus–cancer links potentially direct researchers both 'inward' toward the biomolecular and 'outward' toward the social and cultural, our analysis reveals a predominant shift inward in ways that both reinforce and occlude attempts to banish sexual meanings from view.

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Introduction

In 2005 and 2006, the US public was inundated with advertisements and media coverage relating to a new prevention vaccine targeting the human papillomavirus, HPV, a ubiquitous, although not well-known, sexually transmitted infection (STI), and cervical cancer, a well-known cancer. The case of HPV vaccines demonstrates the intrusion of sexuality into biomedical and health concerns as controversies ignited along several lines, including whether Gardasil might promote 'promiscuity' on the part of teen girls. It then demonstrates the displacement of sexual concerns as pharmaceutical company Merck and Co. pursued a

Table 1: Viruses with cancer connection and known sexual transmission

Year of virus discovery	*Virus/infectious agent*	*Associated cancers*	*Sexual transmission*	*Vaccine introduction*
1964	Epstein-Barr virus (EBV)	Burkitt lymphoma, nasopharyngeal carcinoma, Hodgkin's disease	Possible	No
1965	Hepatitis B virus (HBV)	Hepatocellular Carcinoma (Liver)	Yes	**Yes**
1980	Human T-lymphotrophic Virus I (HTLV-1)	Adult T-cell leukemia/lymphoma	Yes	No
1983	Human papillomavirus (HPV)	Cervical, Anal, Vulvar/Vaginal, Penile, and certain Cancers of the Head and Neck	Yes (primary)	**Yes**
1984	Human herpesvirus type 8/ Kaposi's Sarcoma-Associated HerpesVirus (HHV-8/KSHV)	Kaposi's sarcoma, primary effusion lymphoma, multicentric Castleman's Disease	Yes (considered primary among some populations)	No
1989	Hepatitis C virus (HCV)	Hepatocellular Carcinoma (Liver)	Yes	No

Source: Zur Hausen (1999); Moore and Chang (2010).

strategy of deliberate *desexualization* of the discourse around its vaccine in order to smooth the path to acceptance, regulatory approval and uptake of the vaccine: strategically promoting Gardasil not as a 'vaccine against HPV' but rather as a 'vaccine against cancer' in women (Epstein, 2010; Mamo *et al*, 2010). In effect, the 2005–2006 campaigns introduced the public to a seemingly new conceptualization of cancer, what scientists had known for almost a century: viruses, including some that are sexually transmitted, hold various degrees of causal connection to the subsequent onset of specific cancers in humans (Krueger *et al*, 2010).

While HPV continues to garner the most public and cancer control attention, there are five known additional viruses that can also be transmitted sexually and are causally associated with various cancers. These include: Epstein-Barr virus (EBV); Hepatitis B virus (HBV); Human Herpesvirus Type-8 (HHV-8) also known as Kaposi's Sarcoma-Associated Herpes-Virus (KSHV); Human T-cell Lymphotrophic Virus type-1 (HTLV-1), and Hepatitis C Virus (HCV) (see Table 1).[1] Together with HPV, these are believed to account for about 12 to 18 per cent of all human cancers worldwide – about 2 million cases annually (or 1 in 6 cases) (Hildesheim, 2013), but twice that percentage in low-income countries, or approximately 1 in 4 cancer cases (Krueger *et al*, 2010, p. 3; Schiller and Lowy, 2010, p. 24; Hildesheim, 2013). When including all cancers resulting from weakened immune systems brought on indirectly by HIV and specifically the development of AIDS, the number of cancer cases attributable to viruses increases further.

1 Other infectious agents, as well as parasites and bacteria (for example, *Helicobacter Pylori*), are also linked to cancers, and some are transmissible person-to-person; in the absence of evidence of *sexual* transmission, we do not consider them here.

In this article, we suspend the case of HPV, a case that has received a great deal of attention from scholars as well as the public,[2] to highlight a broader story of entanglements among biomedical research on infectious causes of disease, and ideas about sex, sexual identities and sexual cultures. Through these five other cases of what we call 'virus–cancer connections', we seek to understand how contemporary assertions of a causal link between sex and cancer shapes, how we come to *know* and *prevent* various cancers, and specifically, how these efforts draw on symbolic associations forged between disease risk and sexual morality, practices and identities.

Our analysis begins with the assertion that knowledge of sexual transmission at once directs scientific attention 'inward' to the molecular level of the agents causally linked to cancers and 'outward' to the worlds of sexual practices, cultures and identities that may be causally linked to the risk of infection. We draw on sociologist Shostak's analysis of a different biomedical domain – the study of gene–environment interactions – as a powerful example of how new expert discourses locate health risks simultaneously deep inside the body and out in the external world (Shostak, 2003). We extend this assertion by arguing that – especially because of how sexual practices are implicated in scientific knowledge – these five cases (together with, yet distinct from HPV) straddle the typical explanatory divide between biological/endogenous and social/environmental explanations of cancer causation. Similar to the gene–environment interaction in Shostak's scholarship, the biomedical linkage of the 'inner' molecular world and the 'outer' social world in these cases creates an opening – both epistemic and sociopolitical – that may ultimately be resolved in one of two ways: it may direct the attention of scientists and public health experts to the broader social, political and cultural arrangements that structure the risk of exposure, or it may occlude the social through a narrow and reductionist emphasis on molecular processes alone (Shostak, 2003, p. 2337).

We draw data from biomedical, basic and social scientific, and popular literatures on each viral cancer case beginning with the year the virus was isolated. Drawing on literatures around each case, we examine the places and characteristics in which sexuality – specifically, ideas about sexual practices, identities and cultures – are highlighted, implicated, or banished from view. We pay attention to how expert discourses locate health risks deeply inside the body and/or out in the external world where bodies, lives and the conditions of their existence intertwine. The relative significance and attribution of sexual transmission not only directs our analysis toward sex and sexuality, but also toward the ways this transmission claim might amplify or obscure the arrangements that structure risk and produce vulnerabilities.

Our analysis revealed that the recent encounter between sex and cancer has unfolded in the large shadow cast by the scientific and cultural dimensions of the HIV/AIDS epidemic. Not only do the trajectories of cancers and HIV/AIDS crisscross (Mukherjee, 2010), but the HIV/AIDS epidemic profoundly shapes our understandings of, and the material unfolding of, viral cancer knowledge. HIV/AIDS politics reveal the ways science can

2 For analysis of gender and sexual politics in HPV vaccine introduction see, for example, Casper and Carpenter (2008); Carpenter and Casper (2009a, b); Towghi (2013); Murphy (2012) and so on (Lippman *et al*, 2007; Lippman, 2008). See Wailoo *et al* (2010) for analyses of the political, cultural and social implications of the vaccine's introduction. For a comparison of HPV and HBV sociotechnical trajectories see Mamo and Epstein (2014).

be "held back" (Rosengarten, 2009) by, and deeply entangled with, cultural assumptions about bodies, behaviors and lives. The characterization of an unknown illness as a 'gay disease,' symbolically linked with supposedly excessive and risky sexual practices and the so-called gay lifestyle, highlighted prevailing public and scientific mythologies about the sexual practices of gay men and about sexuality more generally (Patton, 1985; Epstein, 1988; Patton, 1990; Epstein, 1996; Treichler *et al*, 1998). While STIs have varied cultural histories of their own, revealing profound social anxieties and cultural assumptions, HIV/AIDS gave rise to specific cultural linkages of risk, sex, disease and stigma. Our analysis, therefore, is organized in three stages, beginning with 'Pre-AIDS' (c1958–1980), and then the early years of 'the HIV/AIDS epidemic' (c1980–1990s), concluding in the Millennial present (c2000–present).

In recounting this history, we deploy the concept of *sexualization* to refer to the varied places, processes and attributions that infuse a health domain, in this case virus–cancer connections, with implicit or overt sexual meaning and *desexualization* to refer to parallel efforts to banish sexual meaning from view. In specifying these processes, our goal is to understand how scientific assertions, epidemiological constructs and biomedical and public health practices invoke, and thereby produce, conceptions of risk, risky behaviors, and at-risk embodiments and identities that, in turn, attach to certain bodies, practices and identities rendering some hypervisible and others "undiscussable" (Epstein, 2010). Given that sexual contact is a potential means by which many human viruses can be transmitted, we are not surprised to find some degree of sexualization in these cases. Our point is to emphasize how they do so as well as the ways such assertions travel – particularly in ways that, as with HIV/AIDS, variously evoke symbolic associations between sex and cancer.

Sexualization, however, is often met with consistent and powerful efforts to de-sexualize the domain (as was the case with HPV). Cancer researchers, public health experts and corporate actors seek desexualization for a variety of reasons: because sexually transmitted infections are stigmatizing, because these particular infections often disproportionately affect already stigmatized or vulnerable social actors and communities, in response to fears of wading into 'controversial' political matters and prompting a public backlash, or simply out of a discomfort with overt discussion of sexual matters.

We argue that the sidestepping of the sexual matters is simultaneously an evasion of the social and, hence, a reinforcement of a biological approach to understanding and intervening in complex pathways of disease causation. The conditions in which people are made vulnerable to the risk of transmission (for example, discrimination, poverty) *and* to the persistence and oncogenic effects of the viruses themselves (lack of health care, immunosuppression) are often obscured by molecular approaches to cancer research. Thus the simultaneous look 'inward' and 'outward' does not guarantee the development of a 'hybrid, combinatory fields of inquiry'; instead, the biological can occlude the social in ways that erase the structural and interpersonal aspects of health. In short, an optic of sexual risk in this arena of cancer control marks something *new* in the politics of cancer: misplaced ideas of morality, blame and shame in the form of 'risk designations', raising the specter of erasing, sanitizing and eclipsing important social and structural factors in the distribution of health and illness.

Material Matters

The knowledge of specific and known infectious agents as *direct (or indirect)* causes of cancer is significant to the life sciences, biomedicine and public health:[3] if one can control or prevent infection, it is presumed, rates of cancers can be reduced. Yet, neither viruses, nor knowledge of them, are uniform in their materiality or material expression. In the cases we consider, the viruses are, of course, physically different from one another.[4] But, in addition, there are demonstrable differences in the mechanisms of viral transmission; their manifestation in disease, including cancer; and the burden of resulting disease.

First, knowledge of sexual transmission, as well as the actual pathways of this intimate route is uneven and its relative significance differs across cases. Some are transmitted almost exclusively via bodily fluids such as genital secretions (for example, HPV, HCV, HHV-8/KS) or through saliva within or outside sexual conduct (EBV and KS); while others are blood born and associated with perinatal, and needle transmission (for example, HBV, HTLV-1) and implicated in some sexual practices (for example, forced sex, anal and vaginal fisting). The relevance of sexual transmission varies regionally – for example, in the case of HBV, vertical transmission (mother-to-infant) is most significant in HBV endemic countries (for example, regions of Southeast Asia and Sub-Saharan Africa) and needle risk and sexual transmission most significant in non-endemic countries (for example, the United States). And, some viruses are systemic in bodily fluids, including blood (for example, HBV) while others are surface viruses located at the epithelium and affecting associated surface regions such as the cervix, anus and throat (for example, HPV).

Second, the types of infections associated with viruses, and their rates and expression differ. While most are relatively ubiquitous, some clear on their own while others (EBV, KS, HTLV-1) endure in 'healthy carriers', able to reactivate and transmit infection following a period of latency. Some infections, once expressed, can be acute and cause debilitating chronic diseases (for example, chronic cirrhosis of the liver associated with HBV).

Third, the carcinogenic effects of many of these viruses (for example, HCV, KS) are often amplified by HIV-induced immunosuppression, especially, in those individuals who lack antiretroviral treatment. HIV is an indirect cause of what are today known as AIDS-related cancers (it is also the known *direct* causal agent for acquired immunodeficiency syndrome, AIDS). Although HIV is not necessary for cancer to be induced, it holds material (as well as symbolic) significance for how cancers manifest and the contextually varied clinical practices that follow.

Fourth, although these virus–cancer cases are not part of an unknown or devastating epidemic, many of the resulting cancers (whether AIDS-related or not) are devastating illnesses in their own right. The vast majority of those affected are people in low and some middle-income countries, where antiretroviral HIV therapies as well as routine health care and cancer care are less available (Livingston, 2012). Epidemiologically, many of these cancer expressions constitute not only a significant cancer rate (for example, HBV) but also a growing epidemic

3 The term "direct" is italicized to emphasize the scientific understanding that the cellular changes that lead to the transformations that ultimately generate malignancy may be the result of exposure to the agent itself and not an exogenous (environmental) pathway. A direct cause may be a necessary and/or sufficient cause: A virus may be a necessary condition, even if most people infected do not develop the cancer.

4 Viruses differ in their material make up; research continues to understand their properties and expressions.

of coinfection with HIV (HCV, HHV-8/KS). In some cases, such as HTLV-1, while cancers are quite rare, their mortality rates are extremely high. Some viral cancer associations continue to prompt controversy over whether the resulting illness is cancer at all (KS). Cancer expression associated with the viruses and their relative burden varies. Chronic HBV is connected to a small 10–15 per cent of Hepatocellular Carcinoma cases, a form of liver cancer, worldwide (but 50–80 per cent in chronic HBV-positive individuals) and, a very small 1–5 per cent of people with HTLV-1 form cancers (yet the lymphoma cancer cases are known to be 100 per cent resulting from HTLV-1).

There are also important material similarities in these cases: most of the viruses clear on their own in the absence of immunodeficiency; most can be asymptomatic in their hosts; and most are necessary but insufficient causes of the cancers they produce. Many are objects of basic and pharmaceutical vaccine research and development, and in some cases (such as HCV) competition to market is underway. Aside from the HPV vaccines, a vaccine against HBV was developed in 1980 (targeting, not cancers, but diseases of hepatitis more generally). Although these six cases, if we include HPV, may be more different than similar, and although the precise ways in which these oncoviruses intersect with discourses of sexuality varies, what binds them together, we argue, is the ways they constitute an emergent arena of infection and cancer that is expanding in significance. Through an analysis of these five oncovirus cases, we argue that a new sexual politics of cancer has emerged in the 'shadow' of the HIV/AIDS epidemic.

Tracing Sexual Politics: Oncoviruses from Pre-AIDS to Millennial Biopolitics

The sexual politics of our five viral cancer cases spans the 22-year period that begins with what we call 'pre-AIDS' and extends through our millennial present. However, speculation regarding links between sex and disease, and sex and cancer, has a longer history. It was in 1842 that Italian surgeon Rigoni-Stern proposed a causal link between sexual activity and anogenital cancers, arguing against the then-held belief of a common biological origin shared by all cancers (Aviles, 2015). In 1911, Peyton Rous isolated a cancer-causing virus in animals, the Rous Sarcoma Virus or RSV (Zur Hausen, 2006, p. 2), asserting horizontal transmission, in this case from bird to bird, and providing conceptual proof of possible human-to-human transmission, including sexual transmission. This assertion of endogenous cancer causation fueled not only research into viral causes, but also a new line of prevention aimed at inoculating against viral transmission itself. Once viruses (as well as bacteria, fungi, protozoa and so on) were isolated and understood as external pathogens, hypotheses emerged about the role of 'outward' social conditions and behaviors in disease etiology. With the rise of epidemiology, new approaches were joined to molecular optics to examine the interactions among behaviors, environments and biological changes as a means of inferring etiology.

Sexual assumptions endured as part of research into disease etiology, and in the realm of cancers, especially among women. In the late nineteenth and early twentieth century, studies demonstrated a protective role of celibacy in uterine cancers among nuns, prompting the assertion that sexual activity among the married predisposed them to higher rates of cancers (Aviles, 2015). Marriage and religion served as epidemiological proxies for biological

processes related to sexual behaviors (Rotkin, 1973; Proctor, 1995; Löwy, 2010b; Prescott, 2010). While rising rates of this "dread disease" (Patterson, 1987) were linked to various other endogenous social attributes (for example, urbanization, diet and sedentary lifestyles), sexual roles and 'morals' remained objects of focus (for example, sexual abstinence, 'unbridled sexuality' [later termed promiscuity], overzealous breastfeeding, maintaining a well-balanced maternal and sexual life, and safeguarding the organs of maternity were each and all objects of research (Patterson, 1987; Proctor, 1995; Nolte, 2008; Wailoo, 2011, p. 15). The 'race' associated with non-whites gradually became a cancer 'risk factor' as well for the ways it was presumed to determine behaviors (Wailoo, 2011), including sexual ones. And racial classifications intertwined with gender and class in ways that increasingly carried a stigma of sexual immorality (Mei, 2009). Jewish women's "sex hygiene" was associated with low rates of cervical cancer; working class women's high birthrate associated with higher rates; and adolescent and young adults girls with "broken marriages" or out of wedlock births linked to higher rates (Prescott, 2010). Lifestyle – meaning marriage, reproduction and sexual behaviors – were linked to cancer onset while "sexual transmission and its many biological uncertainties" were masked (Braun and Phoun, 2010, p. 47). These theories followed old narratives of poor women's sexual promiscuity and its association with disease as punishment for sexual transgressions (Löwy, 2010a).

More direct associations between women's cancers, viruses and sexual promiscuity are also not new, dating back to at least the mid-twentieth century. For example, mid-twentieth century studies of WWII veterans suggested that sexual transmission of genital warts during sexual activity overseas and later transmission to US wives (Barrett *et al*, 1954) was highly implicated in women's reproductive cancers. Vietnamese women's 'wartime vulnerability' to sexual coercion and rape was associated with high rates of cervical cancer. Genital warts as well as other sexually transmitted diseases (STDs) occupied special significance in the post WWII years. The language of STD held great moral weight given its association with sex and the many ways sex has been an object of campaigns for virtue, rightness and the establishment of codes of conduct and regulation (Friedman and Shepeard, 2007).[5] By the 1960s and into the 1970s, sexual transmission was firmly established as a possible cause of various cancers with understandings deeply intertwined with racialized and class specific assumptions about sexual practices.

Unlike cancer control, controlling infectious disease by targeting bacterial infections and thus its human-to-human spread had seen some success, for example, in cases such as smallpox and diphtheria. The first vaccine against a virus – the polio vaccine, introduced in 1952 – ignited hope that other viral epidemics might similarly be prevented (Heller, 2008). The assertion of an endogenous, known, cause of cancer argued by Rous helped expand this line of cancer research, and, as new technical processes allowed for the isolation, identification and genetic sequencing of viruses by their DNA or RNA, researchers set out to identify other viral cancer cases, including human ones, and new ways to prevent cancers. Hope was realized first with the 1980 introduction of a vaccine against the HBV, known to cause liver cancer (as well as other diseases), and then in 2006 with the HPV vaccines, Gardasil and Cervarix,

5 STDs denote that more than 25 infectious organisms transmitted through sexual activity, along with dozens of clinical syndromes they cause (Eng and Butler, 1997). The term has been shown as symbolically associated with words like "promiscuity", "infidelity", "shame", "divorce" and "embarrassment" (Friedman and Shepeard, 2007).

designed primarily to prevent cervical cancer. These biomedical innovations, we argue, conjoin with these histories of suspected associations between cancer and infectious agents, and between cancer and stigmatized sexualities to demonstrate significant precursors to what we are calling the new sexual politics of oncoviruses that follows. While the case of the recent association of cervical cancer with the oncovirus, HPV, has re-energized and animated a sexual politics that was there all along in different form, this is not the case for the 'other' oncoviruses (Hepatitis B and C, the Epstein–Barr Virus, HTLV-1 and KSHV/HHV-8). Our analysis reveals that what makes these other cases so interesting is the ways and places that sexuality enters and the degrees to which sexuality is tamed.

'Pre-AIDS' (c1958–1980): Infectious cancer and the rise of molecular biomedicine

The first scientifically established *human* cancer virus traces to research in 1958 by Irish surgeon Burkitt while researching an aggressive tumor syndrome affecting children across sub-Saharan Africa (Burkitt, 1958). Burkitt believed the lymphoma was being transmitted by a virus and suspected an association with environmental conditions (rain and hot temperatures). The cancer to which it was linked was later termed Burkitt lymphoma. This same year, the National Cancer Institute of the US National Health Institute launched the "US Virus Cancer Program" and began a systematic search for human cancer viruses (Kuper *et al*, 2000, p. 172; Creager and Gaudillière, 2001; Baker, 2004; Pappas, 2009, p. 962; Yi, 2011). In 1964, virologists in the United States, working with Burkitt's finding, isolated the virus and named it EBV (Epstein *et al*, 1964; Krueger *et al*, 2010, p. 4). While sex and sexuality were not part of this early emergence of EBV cancer, nor its original framing, sexualization processes gradually unfolded outside of the African context. It was its causal link with another disease – infectious mononucleosis (mono) – uncovered in 1967 that launched a mild sexual framing. Researchers Werner and Henle at Children's Hospital in Philadelphia were exploring illnesses caused by EBV and accidentally stumbled upon mono after a lab technician's illness (Miller, 2006). This linked the virus to the so-called 'kissing disease'. (Eventually theories of EBV sexual transmission via genital secretions would further sexualize the field.) Associations of young people and their early sexual experiences with kissing, 'necking' and making out were especially evident in the United States, where heterosexual, mostly white adolescent and young adult men represented the face and object of mononucleosis and its public health prevention campaigns. Pictures of adolescent boys with captions such as "Reaches First Base" and the word mononucleosis were common disease depictions as adolescent health and college health campaigns began to focus less on TB, the "white plague" and more on infectious mononucleosis (Prescott, 2000). This mild sexual framing would endure at least until HIV when a far more urgent prevention campaign came to dominate US prevention efforts.

By the 1960s, cancer was on the cusp of reconceptualization from an unknown "emperor of all maladies" to an infectious disease on the brink of "cure" (Mukherjee, 2010). Findings about EBV informed the 1962 *Life* magazine cover announcing, "New Evidence That Cancer May Be Infectious". The term 'infectious' resuscitated earlier ideas about cancer as contagious and infectious, and also raised hopes of vaccine successes given in the context of the new polio vaccine (Rosenfeld, 1962). In 1964 the *Science News-letter* claimed that a cancer vaccine was

possible (Editor, 1964) and a World Health Organization report asserted a theory of viral cancer causation in animals, appealing for increased federal research monies.

In 1965, The *Journal of the American Medical Association* published the first article reporting on another virus, the 'Australian Antigen', later renamed the HBV. HBV was first isolated in a laboratory by Dr Baruch Blumberg (his colleagues would later identify HBV in blood tests developed to locate the virus in humans, leading to an HBV vaccine). Brumberg was awarded a 1976 Nobel Prize for this research. In 1971, President Nixon declared a 'war on cancer', the National Cancer Act passed, and viral cancer research was funded at high levels for the next decade. Expert and lay communities alike were optimistic that understanding, treating and preventing cancers would see major advances in the near future.[6]

Meanwhile, HBV became the object of overt sexualization. Although absent in the first article reporting on the 'Australian Antigen', sexualization unfolded first with theories of salivary transmission (as well as through semen, vaginal fluids and breast milk). Research included a focus on 'high-risk' sexual and non-sexual practices (multiple sexual partners and IV drug use) in HBV and foreshadowed what would emerge as 'risk group' designations in the 1980s around HIV. In 1971, a blood test was developed for HBV and, in the United States, blood banks began screening donations and testing people for the presence and persistence of the antibodies that detect the virus. Blood screening research led to the eventual knowledge that the infectious agent itself can produce an immune response and, therefore, was a prime candidate for the development of a preventive vaccine. Gay identified men, who had higher rates of HBV, were prompted to volunteer to serve as early human subjects in HBV research and help in the development of the vaccine. Throughout the 1970s, researchers and plasma centers forged partnerships with gay organizations, what were then called venereal disease screening clinics, to recruit 'homosexual' men for vaccine research trials (Editor, 1980). Such activities paved the way for rhetorical associations between risky sex, gay men, and the HBV.

The discovery of the retrovirus known as Human Lymphotropic Virus Type-1 (HTLV-1), isolated in the laboratory of Robert C. Gallo (who later became known for his work on another retrovirus, eventually termed HIV), marked another important convergence between sexual transmission and cancer. In the 1970s, Gallo had been working at the NCI pursuing evidence for his conviction that viruses cause human cancer. The cancer, Adult T-cell Leukemia or ATL, was first proposed as a disease entity in 1977 in Japan, as researchers described geographic regions with a high incidence of T-cell-associated lymphoproliferative disorders that may have had a viral origin. In 1979, Dr Poiesz, Ruscetti, and co-workers isolated HTLV-1 from a T-cell line in a patient with cutaneous T-cell lymphoma (Poiesz *et al*, 1980; see Gallo, 2005 for a detailed history), thus identifying the first known human retrovirus as a causative agent for human malignancy (International Agency for Research on Cancer, 2011, p. 323). Researchers in France also were also pursuing this conviction, identifying the role of several human T-cell leukemia retroviruses in causing leukemia and lymphomas. Such etiological claims in cancer were driven by molecular biology and, specifically, oncogene research (Fujimura, 1988; Gaudillière, 1998). As sociologist Fujimura has described, a "bandwagon" of molecular research into cancer emerged as a result of studies into viral oncogenes.

6 In 1980, NCI leaders broke up the VCP and integrated the pieces into other NCI programs (Rettig, 1977), with negative effects on viral cancer research funds (Fujimura, 1988).

Two lines of inquiry exemplified this 'Pre-AIDS' period: first, scientific inquiry into causes of disease at the molecular and sub-molecular levels, including causes of cancer, and second, research into associations between sex and disease, based on analyses of bodily fluids and transmission practices, some of which were sexual in nature. These simultaneous ways of apprehending cancer and doing cancer prevention – an 'inward' attention to cancer that includes identifying a *biomolecular agent* (a virus) as an actor in the etiology of disease with its associated biomedicalization processes,[7] and an 'outward' view toward people's behaviors and environments – provide early evidence of the ways sexualization processes unfold in viral cancer knowledge. To be sure, the overt speculations linking sex and disease gradually diminished with the rise of molecular biology and epidemiology – a process we call desexualization. But overt sexual framings would re-emerge with the onset of the HIV/AIDS epidemic.

The emergence of HIV/AIDS (c1980–2000)

In the standard historiography of AIDS, the disease is often said to have entered public discourse with the publication in the *New York Times* on 3 July 1981 of a short article, by medical reporter Altman (1981), entitled "Rare Cancer Seen in 41 Homosexuals". The "cancer" referenced was Kaposi's sarcoma, the purple blotches that had appeared on the skin of young gay men in New York and California. Previously, KS typically was known to affect elderly men of Mediterranean descent, among whom it was not terribly aggressive, although it also was endemic in sub-Saharan Africa. Yet neither of these KS conditions garnered much public discourse nor associations with sex and sexuality.

The appearance of KS in a group of severely ill young men in the United States, alongside other conditions not normally found in that population, was the first indication that something new and troubling was afoot (Epstein, 1996). And from the outset, that troubling character was discursively associated with a dangerously excessive sexuality. Perhaps because KS, with its distinctive lesions, was much more visible than other conditions associated with AIDS – indeed, perhaps because KS lesions functioned, in effect, as the stigmata of a deeply stigmatized condition – this cancer quickly became the telltale marker of the new syndrome in Western countries, as well as its metonymic stand-in. Before the invention of its official acronym, many described the new health threat as "the gay cancer" (Epstein, 1996). While problematic in its characterization of the condition as being somehow intrinsically 'gay', the phrase alerted gay men to the presence of a significant new danger and constituted early organizing efforts (Epstein, 1996).

In the early years of AIDS, science seemed to be invested in an already present moral claim about 'good' and 'bad' sex, and 'moral' and 'immoral' sexual identities. Treichler (1999) later referred to AIDS as an "epidemic of signification" for the ways that the discourses surrounding it linked the disease with both sex and stigmatized sexual identities. While viruses are transmitted through bodily fluids, not by categories of people, HIV/AIDS discourses seemed to divide people into moral terrains with some bodies and lives mattering more than others, not as objects for medical treatment, but as objects of stigma and sites of contagion. The discrimination associated with same-sex behaviors that may have shaped risk

7 Biomedicalization is a term used to capture broad technoscientific innovations that shape shifts in biomedicine, including the "molecular gaze" and "vital politics" (Clarke *et al*, 2003; Rose, 2007; Clarke *et al*, 2010).

of transmission was obscured from scientific understanding in favor of continuing shame and blame. These sexual politics, which have been widely documented, gradually faded in intensity. The cancer-fighting drug AZT (approved by the FDA in 1987 to treat people with HIV/AIDS), while toxic and expensive, began to diminish the overwhelming mortality of HIV and its sexual politics. Therapeutic 'cocktails' or highly active antiretroviral therapy (HAART) came later in 1996, further shifting the contours of the epidemic and desexualizing its attributions, although its ability to symbolically cling to the bodies of men who have sex with men (MSM) has not been banished from the view. Many people lack treatment in the United States, and the vast majority of people living in low resource countries have limited access. Pharmaceuticals, and their politics, continue to shape the contours of the HIV/AIDS epidemic and are significant for what is emerging as an HIV–cancer complex.

Despite the initial prominence of KS as a signifier of AIDS, over time this cancer gradually began to lose its capacity to stand-in for, or carry the metaphorical weight of, the AIDS epidemic – even as sexual politics continued to shape its discursive and material history. For one thing, it became apparent that the epidemiological spread of KS was not coextensive with that of AIDS: KS was more likely to be found among MSM than other groups at risk of AIDS (De Jarlais *et al*, 1984; Haverkos *et al*, 1985; Mortimer, 1987). In addition, beginning with the discovery of HIV in 1983, AIDS itself gradually came to be defined in relation to the effects of HIV in destroying the immune system, rather than being characterized solely by the opportunistic infections and cancers that followed. And, with the advent of HAART in the mid-1990s, KS itself began to vanish from public view, at least in places wealthy enough to provide the drugs to its citizens.

While narratives of KS and AIDS began partially to diverge, the new biomedical salience of KS led researchers to invest in understanding its etiology. Researchers began to suspect that KS was caused by yet another virus – one that rarely causes disease, unless its host is immunosuppressed. That KS was so much more prevalent among MSM with AIDS than among AIDS patients with hemophilia led scientists to speculate that a distinct sexually transmissible infectious agent might be at work. In 1994, Yuan Chang (a pathologist) and Patrick S. Moore (an epidemiologist) isolated DNA fragments from a KS tumor in an AIDS patient and published an article in *Science* reporting what they labeled "KS-associated herpesvirus-like (KSHV) sequences" (Chang *et al*, 1994). As evidence accumulated about a causal link of a virus with KS, the abbreviation KSHV was adopted for the virus itself, known as Kaposi's sarcoma-associated herpesvirus. Subsequently, however, the International Committee on the Taxonomy of Viruses renamed it the human herpesvirus 8 (HHV-8), partly for reasons of consistency in naming, and partly because of its apparent link with other rare cancers besides KS, including multicentric Castleman's disease and primary effusion lymphoma (RNCOS, 2012, p. 375).

Meanwhile, in the early years of the AIDS epidemic, HBV underwent renewed sexualization insofar as it took on parallel 'risk group' designations to that of HIV/AIDS. In 1981, an inactivated vaccine was approved, followed by the approval in 1986 of a DNA synthetic vaccine (Hepatitis B Foundation, 2009). Though gay men were largely to thank for the research discovery, such reference was rare in mainstream media coverage of this vaccine miracle (Conis, 2010). A link to gay sexuality as well as IV drug use shaped the framing of the HBV vaccine as a controversial one against an STI found among highly stigmatized groups. Throughout the 1980s in the United States, public health advocates worked to desexualize

(and destigmatize) the disease, Hepatitis, and the HBV vaccine by effacing the causal and symbolic assertions of sex, drugs and disease. Particularly in light of the emergence of the HIV epidemic and its symbolic associations with stigmatized sexualities and practices, such distancing from sex and sexuality was needed if this disease association and especially its new vaccine would become an accepted part of the health-care marketplace. These efforts would be eclipsed by the 1991 introduction of the HBV vaccine to the schedule of vaccines given to newborns, thereby desexualizing the discourse. However, public scrutiny and a sexualization framing around Hepatitis B would re-emerge at the close of the 1990s with a controversy over the links between vaccination and neurological disease. As historian Colgrove (2006, p. 231) argues, "The animus against the vaccine among critics was rooted … in the fact that it protected against blood-borne disease spread primarily by sexual contact and injection drug use (although transmission also occurred perinatally and among children in day care facilities)". At the US 106th congressional hearing in 1999, one parent activist testified: "Almost every newborn baby is now greeted on its entry into the world by a vaccine injection against a sexually transmitted disease", and goes on to say, "because they couldn't get the junkies, prostitutes, homosexuals, and promiscuous heterosexuals to take the vaccine" (Colgrove, 2006, p. 231).

As such concerns eventually subsided, HBV vaccination became fully viewed as a legitimate component in a large-scale, newborn immunization program and as a success story in cancer prevention. It would, therefore, seem that sex is fully tamed in this case. Yet, because the vaccine is not 100 per cent effective and may not provide lifetime protection (Centers for Disease Control and Prevention, 2010), other precautions such as the use of condoms and other barrier methods during sex and not sharing needles are recommended health prevention tools. Concerns about coinfection with HIV and HBV have further reinforced the associations between HBV and such forms of behavioral prevention. Therefore, certain symbolic associations among sex, health and morality continue to be found beneath the surface of public discourse about Hepatitis B. The case therefore nicely suggests the practical limits of desexualization, even in a case where therapeutic triumph would seem capable of displacing sexuality from view.

Finally, HTLV-1 is also deeply entangled with the HIV/AIDS epidemic. Often referred to as a forgotten human retrovirus lost in the wake of attention to HIV,[8] HTLV-1 infection was found in high rates among drug users; it occurs as a frequent coinfection with other HTLV viruses and with HIV. Of the five oncovirus cases we consider, HTLV-1 is the one in which the link between a sexually transmitted viral infection and cancer is least well-developed. The virus is primarily transmitted vertically from mother to infant (likely via breastfeeding), and because this is the most feasibly preventable route, it has been the most intensely studied (Yamaguchi, 1994; Krueger *et al*, 2010, p. 475). Ultimately, of the possible viral reservoirs, "breast milk and blood have both been well-established […]; blood appears to be the most efficient medium of infection" (Krueger *et al*, 2010, p. 475).

Nonetheless, extensive research investigates the sexual transmission of HTLV-1 and its prevention possibilities, with a majority of research conducted with either 'sex workers' or patients at STD clinics. Research, thus, often includes 'sexual risk group' designations and

8 HTLV-1 began with its discovery in 1979 in Gallo's NCI laboratory; Gallo's designation of the virus linked to AIDS as "HTLV-3" did not "take".

reveals high coinfection of HIV and HTLV-1. In Latin America, HTLV-1 is considered an STD of high importance, with the virus found in genital secretions, and with vaginal and anal intercourse as important factors for transmission (Tajima *et al*, 1982; Gotuzzo and Verdonck, 2004). Research focuses on sexual transmission and 'safe sex' prevention strategies emphasizing condom use (in part because of questions surrounding specific modalities of transmission). Condom use as a prevention strategy is found in most documents related to HTLV-1 and is strongly recommended for those "with multiple sex partners or otherwise engaging in non-mutually monogamous sexual relationships" (Gotuzzo and Verdonck, 2004, p. 452). Some sources explore the degree to which cultural and gender differences might affect sexual and contraception practices in particular areas of endemicity (International Agency for Research on Cancer, 2011, p. 325). Overall, the prevention paradigm seems to be one of general 'safe sex' with one exception of treatment as prevention in the case of cervicitis among Peruvian sex workers (Zunt *et al*, 2002).

In short, during the final two decades of the twentieth century, several virus–cancer cases took shape against the backdrop of the research trajectories and sexual politics that characterized the HIV/AIDS epidemic. Signaling a new era, in 1999, an Institute of Medicine report titled "The Hidden Epidemic" (Eng and Butler, 1997) called upon government and private organizations to support STD prevention activities as a strategy to prevent "STD-related cancers", asserting that secrecy and stigma around STDs is contributing to lack of efforts around these infections (excluding HIV). That same year a benchmark study of HPV would posit sexual activity as a cause of cancer (Walboomers *et al*, 1999) and usher in a new period marked by intense concern with virus–cancer connections.

Millennial present c2000–Present: The global governance of cancer

The Millennial Present (c2000–2015) is marked by significant changes in the economics, technoscience and organization of biomedicine (Clarke *et al*, 2010). In the United States, biomedical research and clinical care have been reshaped by disease awareness campaigns, risk-reduction approaches, direct-to-consumer-advertising, and 'patient participation' across the world-wide-web. Throughout the globe, widening economic inequality produced by neoliberal politics and its reliance on privatization and public–private collaborations are reshaping health-care infrastructure, biomedical research and pharmaceutical R&D (Geissler, 2015), as well as new formations of government and new modes of collectivization (labeled "biological", "therapeutic" or "pharmaceutical" citizenship) (Ecks, 2005; Rose, 2007).

Large-scale population level policies and practices, including immunization guidelines, represent new modes of biomedical intervention, producing and protecting certain bodies and lives in the name of health. As anthropologist Murphy (2012, p. 14) observes, "Population is one aggregate materialized among many others that unevenly enacts biopolitics", referring to the stratification of group-based public health prevention approaches. As viral cancer associations are scientifically established, the claims shape in-country health care, international health policy and pharmaceutical R&D. For example, a global alliance for vaccines (the GAVI alliance) formed as a private–public partnership and global strategy to ensure "equal access to new and underused vaccines", including HPV vaccines to combat cervical cancer (www.gavi.org).

The burden of HIV/AIDS and of viral cancers is today experienced by low-income countries (Krueger *et al*, 2010). Structural inequities (water, food and nutrition insecurity; housing

instability; and other mediators of immunosuppression) shape health and illness, as well as rates of HIV, other viruses and cancers. Despite the introduction of HAART in 1996, with resource rich countries seeing the emergence of long-term survivors as a new population of health concern, the vast majority of the world's poor living with HIV do not have access to consistent and immediate life-saving medications. Rates and burdens of HIV/AIDS remain high, as does an emerging epidemic of 'AIDS-related cancers'. The search for an HIV vaccine, as well as a means for current drug therapies to eliminate HIV among those already infected, rely on vast amounts of money and 'partners' linking clinical laboratories, research facilities and pharmaceutical companies.

It is against this backdrop that we describe new developments in relation to our five viral cancer cases. Recent attention appears to be highlighting a possible link between EBV and 'AIDS-related' lymphomas. Here, the association linking EBV and cancer includes complex entanglement with HIV and immunosuppression. A direct cause in the case of EBV is less clear: Only about 20 per cent of cases of these lymphomas in the developed world are associated with EBV. Given the correlation with immunosuppression and specifically its 'AIDS-related' designation, an incipient sexual framing may renew as associations with sexual practices circulate around HIV.

Yet, what characterizes EBV cancer knowledge is regional variation, specifically its association with poverty.[9] Owing to lack of cancer's visibility as a major health issue in many low-income countries (where malaria and HIV are most visible and the fight against them most funded), EBV and EBV-associated cancer knowledge are low. What is known is that where EBV-associated cancers are most prevalent, EBV transmission is predominantly via saliva and occurs in the first years of life (with one's mother), with only some cases delayed until adolescence or young adulthood.[10] Salivary transmission in the context of mother–infant connection is not a site of sexualization but one of maternal child health. Thus, EBV is not associated with a sexual frame in low-income countries. However, it is associated with the development of cancer with 'immunosuppression' caused by HIV as a condition implicated in the pathogenesis from EBV infection to cancer. Research into EBV in HIV-positive individuals shows a 60 per cent higher risk of developing a cancer than among those who are seronegative (Cohen *et al*, 2011). HIV-related (non-Hodgkin) lymphomas represent a significant source of morbidity and mortality among HIV-infected individuals and is the most prevalent AIDS-defining cancer. While rates have decreased, presumably as a result of the advent of HAART, cancer risk remains elevated for people with HIV.[11]

By contrast, in the United States and other high-income countries, salivary transmission of EBV in adolescence continues to receive etiologic attention, and moreover this emphasis on salivary transmission increasingly includes genital-sexual contact such as the use of saliva as a lubricant (Krueger *et al*, 2010). In addition, sexual transmission via semen was documented in a 2007 publication based on research conducted with students at Edinburgh University. Researchers found sexual activity, measured by number of sexual partners and degree of

9 The primary burden of BL lymphoma as well as other EBV-related cancers is in Sub-Saharan Africa, and in some regions of East Asia for nasopharyngeal cancer (Parkin, 2006).

10 Early transmission is associated with developing countries, and delay with developed countries where standards of living are higher (see Higgins *et al*, 2007).

11 Approximately 90 per cent of Hodgkin and immunoblastic lymphomas in HIV-positive people are EBV positive and 50 per cent of Burkitt lymphomas in HIV-positive people are EBV positive (Cohen *et al*, 2011).

condom use, to be a key factor in transmission (Higgins *et al*, 2007). Measuring sexual risk in these particular ways is standard in post-HIV research and contributes to reframing what has long been a muted emphasis on sexual transmission via 'kissing': Now we see a more consistent sexualization produced by a hypothesis of transmission via genital-sexual activity. Yet, by and large, the EBV viral cancer association has not entered public awareness. This, we believe, is because of the enduring knowledge of its well-known non-cancer association with 'mononucleosis', as well as with the scientific uncertainty surrounding its cancer connection.

In the case of HTLV-1, recent developments include a continued mild sexualization as an STI, but significant molecularization and biomedicalization of the HTLV-1–cancer connection. HTLV-1 (like HHV-8 and HPV) is now believed to be responsible for almost all cases of its associated cancer, in this case T-cell leukemia; it is also believed to be a necessary *and* sufficient causal agent. HTLV-1 also represents a major shift in cancer taxonomy resulting from molecular DNA identification: The presence of the cancer itself has come to be equated with the virus itself, making the virus a 100 per cent causative factor in this cancer (Krueger *et al*, 2010). This conceptualization may prove to be an important influence on the direction of STI-cancer associations, especially the ways these shape prevention strategies as the existence of the virus itself, an STI, may come to represent disease. In addition, HTLV-1 (similar to EBV) is emerging as an object of pharmaceutical and biomedical R&D as part of a prophylactic vaccine business model (Lynch and Kaumaya, 2006).

For KSHV/HHV-8, the sexual politics remain subsumed within those that swirl around HIV/AIDS. Today, HHV-8 is believed to spread primarily through saliva. (It can be transmitted in a number of ways, including through blood, sexual contact and organ transplantation.) Researchers have generally presumed the significance of sexual transmission in the case of MSM in Western countries where a distinct sexualization of the discussion continues. Nonetheless, there is little clear evidence about precisely which sexual practices might be implicated. Speculation has focused on practices that involve exposure to saliva, including deep kissing, oral-genital and oral-anal contact, and salivary lubricant in anal sex (Dukers *et al*, 2000; Martin and Osmond, 2000; Butler *et al*, 2009).

Another process of mild sexualization of HHV-8 is found with its link to HIV infection as an important co-factor for many, certainly by suppressing the immune response, but possibly also by promoting tumor growth through other mechanisms (Schiller and Lowy, 2010). Some researchers suggest, "the greatest advances in terms of exposure prevention may involve the promotion of safe sex practices that simultaneously address the transmission of both viruses [HIV and HHV-8]" (Krueger *et al*, 2010, p. 453). Dubbed "a second tier vaccine target", HHV-8 has seen little vaccine research in comparison with other oncoviruses (Schiller and Lowy, 2010). The reason is partly one of simple epidemiology: HHV-8 is linked to only 1 per cent of the worldwide cancer incidence. Moreover, primary infection is usually asymptomatic, and the implementation of effective antiretroviral therapies to treat HIV infections has led to dramatic reductions of KS. Therefore, efforts to prevent or treat HIV may be the best approach to interrupt KSHV infection (Schiller and Lowy, 2010). Yet research in low-income countries shows that the stigma associated with KS lesions as a visible manifestation of HIV infection inhibits people from seeking cancer treatment, as do the usual economic issues (Amerson *et al*, 2013).

In the new millennium, amid reports of an 'epidemic' of sexually transmitted HCV infection among HIV-positive MSM in Europe, the United States and Australia, HCV has become an

interesting case of incipient sexualization via a link to HIV and a symbolic association with 1970s drug and sexual cultures. HCV was identified in 1989 and had become a growing public health concern by the millennium. Although primarily linked to needle-based drug use, perinatal transmission in childbirth from mother to child and through blood transfusions in the earlier twentieth century are the cause of much of the global burden. Recently, however, MSMs have been considered to be at higher risk if they engage in unsafe sex, and some researchers have expressed concern about specific sexual practices such as fisting or the use of sex toys that can damage rectal mucosa (American Association for the Study of Liver Diseases, 2010; Krueger *et al*, 2010, p. 299; Tohme and Holmberg, 2010).

The newly sexualized portrayal of HCV included scattered reports of sexually transmitted HCV among HIV-positive MSM in several northern European countries (Fierer, 2010) in Australia and the United States. In 2008, an article in *Sexually Transmitted Infections* expressed concern about a "possible outbreak" of HCV among HIV-positive MSM in London and Brighton, based on testing performed in clinics from 2002 to 2006 (Giraudon *et al*, 2008). The United States, CDC expanded HCV testing recommendation to persons infected with HIV in 1999 and, a decade later, HIV-linked HCV among MSM was being described as an "epidemic", and indeed one that, despite its "very recent origin", had become "truly international" (Fierer, 2010). Then in July 2011, the CDC's *Morbidity and Mortality Weekly Report* focused on data from New York City from 2005 to 2010: Published in the midst of public attention to the 30th anniversary of the first reporting the AIDS epidemic, the MMWR article observed that hepatocellular carcinoma, typically linked to HCV, had become a leading cause of death among HIV-infected persons in the United States who had not progressed to AIDS, in part because of HIV's effect in accelerating the progression of HCV disease (Centers for Disease Control and Prevention, 2011). Media attention following declared an "explosion" of HCV among HIV-positive MSM: "These men are sitting ducks for liver cancer", said Lynn Taylor of Brown University, "… We are seeing tons of gay men newly diagnosed with HIV, and then with HCV. I could go to a funeral of an HCV patient every week" (as quoted in (DeNoon, 2011, p. 1). Daniel Fierer of New York's Mount Sinai School of Medicine noted the statistical linkages between HCV transmission among MSMs and a variety of risk practices, including unprotected anal sex and methamphetamine use (that can greatly prolong sexual intercourse). Researchers also point to the erectile dysfunction drugs (often taken recreationally) as increasing risk given a prolongation of sex. The pharmaceutical industry and DTC advertising that moved these drugs into popular use, then, are highly significant to processes of sexualization swirling around HCV.

Health promotion messages also participate in this incipient sexualization: As Lenton and Fraser (2016, p. 44) persuasively argue, these resources "figure people living with hepatitis C as intrinsically anomalous (and) run the risk of inadvertently naturalising stigma, anxiety and fear surrounding intimate contact". Based in Australia, the authors urge those engaged in health promotion to "carefully examine the messages they produce if they are to avoid creating uncertainty and anxiety about the implications of hepatitis C for sexuality and intimacy" (Lenton and Fraser, 2016, p. 44).

Sexualization also emerged with research confirming an age cohort of baby boomers (born between 1945 and 1964) designated as at high risk for persistent HCV infection. While the CDC states the reasons for high rates of this age cohort are unknown, public discourse implicated "rock 'n' roll, sexual liberation, and HCV" as an explanatory framework

(Cohen, 2012). Most discussions of behavioral prevention strategies mention interruption of sexual transmission (for example, via condom use) only in passing. In this period of heightened consumer marketing and personal responsibility for one's health, patient information billboards and DTC advertising for Hepatitis C testing as well as Hepatitis C medications promote a 'will to health' for those now in their 50s to 70s for whom cancers are a very real threat. Thus the current sexualization of HCV seems to extend across 'risk groups' and different stages of the lifecourse to encompass both young MSMs and older adults, all of whom are called upon to become responsible for their health.

Discussion: Sexual Politics, Disease Prevention and Sexual Health Promotion

In our analysis of viral cancer connections, we have set aside the better-known case of HPV – and cervical cancer, and its reanimated sexual politics. Our focus instead is on the five lesser-known examples of virus–cancer linkages. With the HIV/AIDS epidemic as crucial backdrop, we examined where and in what places sexual meanings and politics emerge and are subsumed in these cases. We asked: How might a simultaneous 'inward' and 'outward' biomedical and public health lens result in the development of a hybrid combinatory field of inquiry (as Shostak found in the case of epigenetics)? Relying on the concepts of sexualization and desexualization to point us toward the ways attributions of sex and sexuality are bound up with viral cancer knowledge and where they are banished from view, we explored the five viral cases across three time periods. Despite differences in the material characteristics of these viruses and their associated cancers, we found a consistent tendency for evidence of sexual transmission to prompt both familiar and renewed ideas about disease and stigmatized identities. We found that despite differences in the material characteristics of these viruses and their associated cancers, sexualization processes variously unfold in ways that produce familiar and renewed ideas about sex, disease and stigmatized identities.

Overall, HBV is perhaps the case whose trajectory is most similar to HPV in terms of how sexualization processes took hold early with viral cancer knowledge and gradually diminished as prevention technologies entered medical practice. Conceptions of HBV risk groups as stigmatized, others eventually faded as the HBV vaccine entered newborn immunization guidelines as a legitimate component of routine health prevention. Similarly, concerns about HPV and HPV vaccination revolved around the sexualities of teenage girls and panics around their presumed future risk. Such fears were gradually toned down with a strategy of marketing the vaccine not as STI prevention but as cancer prevention. Together, these 'cancer vaccines', reveal persistent processes of desexualization as vaccine technologies are developed and pathways adopted to ensure their use (Mamo and Epstein, 2014). Yet desexualization is uneven and incomplete, especially in the case of HPV. As HPV-related cancer cases rise, declarations of an 'epidemic' of oral cancers emerge, and attention and controversy around anal cancer, especially among 'high risk' populations of HIV-positive men and women, and MSMs, percolate. For HBV recent attention to HIV coinfection has also continued to highlight 'risk practices' in familiar ways. These developments resurrect the symbolic associations between sex, health and morality as stigma appears to cling to certain bodies and practices.

The absence of a vaccine 'solution' in the other four sexually transmitted virus–cancer cases is significant to the shaping of their sexual politics. In the case of HPV and HBV, pharmaceutical success raised the prospect of desexualization, though not without complications and counter-tendencies, as we have described. Moreover, the vaccines' introductions required public acceptance to ensure policy and marketplace uptake, and in practice the power of pharmaceutical companies helped significantly to ensure their success (Mamo and Epstein, 2014). In the other five cases, with the absence of a pharmaceutical approach, we find other patterns. EBV, for example, emerged as linked to a relatively benign kissing disease, but largely underwent desexualization given its ambiguous causal connection to cancer and regional variation. And, the case of HCV, in slight contrast, reveals only an incipient sexualization via its links to HIV and a recent focus on HCV infection as 'epidemic' among HIV-positive MSM and baby boomers. HHV-8 is a case with early intersections with HIV/AIDS and the history of 'gay cancer' and subsequent investigation into specific transmission routes among MSM. Even as KS began to lose its capacity to stand in as a signifier for AIDS with its metaphorical weight of sexual stigma, it has never fully shed its sexual association. Finally, HTLV-1 discourse moved from an initial focus on heterosexual, married couples to research on sex workers and other 'at-risk' groups in low- and middle-income countries. Attention included comparative 'safe sex' practices, as well as continued emphasis on a story of HIV coinfection. EBV and HHV-8/KS (both herpes viruses) seem to follow inverse discursive plots (Krueger *et al*, 2010, p. 393) "HHV-8 [was] first considered to be mainly a sexually transmitted infection (especially among homosexual males), [but] is now also seen to be transmissible by saliva". Conversely, EBV was first associated to a kissing disease transmitted via saliva and is now gaining understanding as linked to sexual transmission and cancer. While the story of HHV-8 and KS continues to fasten on the details of homosexual sex, the sexual practices implicated are by no means restricted to MSM.

The sexual dynamics of HTLV-1 are politically aligned with HIV prevention messages of 'safe sex' and especially condom use and global prevention efforts that largely target sex workers. Peru, for example, adds a focus on HTLV to its HIV prevention strategies, asserting that co-infections are frequent and that risk follows the same routes of sexual transmission. The politics of HTLV-1 and its association with cancer may become increasingly important in the story of STI-cancer connections. Even though Gallo failed in his attempt to link AIDS to the HTLV family of viruses, current research on HTLV-1 takes place in the shadow of the health prevention infrastructure that has grown up around the HIV/AIDS epidemic.

In the case of HCV, a gradual shift is underway from viewing it as a risk particular to injection drug users (given blood-borne transmission) to speculation of a hidden epidemic of HCV in HIV-positive men and women, as well as baby boomers, especially women. Controversial grassroots strategies to reduce HIV risk, such as "serosorting", and new, mainstream public health approaches to reduced HIV risk, such as pre-exposure prophylaxis, are targeted for critique because of presumed potential contribution to an HCV epidemic among HIV-positive MSM (Tohme and Holmberg, 2010, p. 1500). This new focus on both the HCV/HIV link (and its possible contribution to an "outbreak", "epidemic" or "explosion" of HCV infection in HIV-positive MSM) and an HCV epidemic among people born in 1946–1964 is likely to promote attention to sexual practices in general, and gay men's sexuality in particular. Much as in the early years of the AIDS epidemic, it appears that a focus is on those sexual practices that most diverge from mainstream social norms.

EBV, which began as linked to a kissing disease, gradually emerged as a sexually transmitted herpesvirus of great concern for HIV-positive people and those with compromised immunity. The virus is today of growing importance to autoimmune and HIV activists and researchers, the World Health Organization, and vaccine R&D efforts. Developing "cancer vaccines", including one for EBV (and HCV) is considered to be a "a huge opportunity" and the "most promising market" for pharmaceutical vaccine development across the globe, with the United States accounting for 49 per cent market share (RNCOS, 2012). The hype and hope surrounding these economic assertions include therapeutic cancer vaccines as well as those for primary prevention.

Overall, the conjoinment of STI with cancers at times sexualizes basic research, clinical practice and public health approaches and, at times, prompts attempts to banish sex from view. In highlighting sexual politics – or more specifically, how and when sexualization and desexualization processes unfold, we have sought to show that the link to infectious disease and the sexualization of this segment of cancer control marks something *new* in the politics of cancer. We find that discourses of sexuality, when they appear in virus–cancer connections, unfold in ways that re-energize and reveal specific (and sometimes longstanding) cultural linkages of health and disease with risk, sex and stigma, recast in a new language of modern biomedical risk and pharmaceutical possibilities. These developments demonstrate Foucault's (1980) general contention that modern societies are characterized less by simple repression of sexuality than by a proliferation of discourses and practices relating to sexuality, health and the body aimed at managing populations in the service of numerous social ends.

While the specific dynamics of sexualization and desexualization differ across cases, the general pattern is that as STI viral cancer associations are invoked so too are various conceptions of risk, risky behaviors and at-risk embodiments that point outward to social explanations of disease causation. Yet, as these fields of research are transformed through biomedicalization – for example, through the biomolecular approach of the vaccine 'fix' – we find attempts to desexualize public discourse: The assumption is that preventing sexual transmission simply becomes less important. The suppression of sex and sexuality is an ongoing process and one that in these cases has been accomplished only with difficulty, if at all.

While biomedical interest in identifying oncoviruses and their potential vaccines is likely to expand, especially as President Obama's "moon shot" to cure cancer rolls out, cancer control and prevention remains dominated by medical screening technologies and so-called lifestyle risk behavior change.[12] Meanwhile, structural factors shaping intimacies of social life, as well as the full range of the social determinants of health, increasingly remain outside the optic of biomedicine. As the politics of HIV reveals, biomedical 'fixes' are often heralded as the best hope for disease prevention, eclipsing decades of social and behavioral approaches. In the early 1980s, safe sex practices, reducing the number of sexual partners, and other social and behavioral approaches joined education and psychosocial practices to intervene in the social and sexual aspects of people's lives, identities and cultures as a means of preventing illness. At the same time, many publics and some public health communities turn attention to power,

12 Cancer treatment research, in contrast, is dominated by genomics, as well as the ongoing focus of surgery, chemotherapy and radiation. We suspect we will see increased attention to immunotherapies perhaps implicating viruses as therapeutics.

power relations and structural determinants of health, fighting to end inequality, discrimination and disempowerment, as fundamental causes shaping HIV infections and uneven treatment.

Today, it is most often the exposure to the risk of HIV infection – not the social conditions of that risk nor the behaviors *per se* – has become the object of intervention through pharmaceutical chemoprevention drugs and surgical methods (Giami and Perrey, 2012). While HIV is not the only case where risk itself is the object of treatment (for example, the birth control pill (Oudshoorn, 1994); cancer chemoprevention (Fosket, 2010)), targeting potential exposure to the risk of transmission as the object of intervention represents a biomedical turn and an inward conception of disease prevention.

Together these reveal how pharmaceuticals, including 'cancer vaccines', are today shifting cancer prevention from a field with few advances and multiple competing claims to one of biomedical R&D potential. While the HPV and HBV vaccines are the most notable and only biomedical approaches to cancer prevention on the market today, other 'cancer vaccines' targeting viral infectious agents are in various stages of clinical trials and the object of pharmaceutical dreams. When approaching prevention from the perspective of the virus, primary and secondary prevention efforts take on new meanings. Primary efforts could include avoiding exposure, preventing infection once exposed, prophylactic eradication of the infection and preventing cofactors that participate in processes of cancer cell change from non-malignancy to malignancy. Secondary prevention could include any interruption of non-malignant disease due to infection, be it by preventing cofactors or therapeutically eradicating the viral disease itself. The usual 'secondary' prevention approaches of screening and treating cancer would be joined by this viral approach.

However, the viral cancer linkage that we have taken as our starting point directs us toward the material-discursive connections forged between viruses and disease. There remains uncertainty that often goes unnoticed: Some cancers are formed not from exposure to the virus through sex, but from the persistence of viral infections that either remain asymptomatic or persists in its expression yet remains untreated because of economic, political and biomedical infrastructures and processes. As Löwy (2010a) argued in the case of cervical cancer in Brazil, theories of cervical cancer followed old narratives of poor women's sexual promiscuity and its association with disease as punishment for sexual transgressions (Löwy, 2010a), yet over time "the tropical scourge" of cervical cancer revealed not "lifestyle" or "morality" but the structural conditions of poverty (and its associated poor hygiene) that places women at risk of viral persistence. Further, while some cancers may develop as a result of past sexual transmission and latency periods, the vast majority of human cancers remain unknown in their etiology. This framing serves to shift attention away from unknown social-environmental causes of cancers, explanations that remain largely understudied.

Our analysis reveals, then, that in the spaces between viruses and cancers and in their very interaction lies sociocultural *uncertainty* about the impact of 'upstream' political, social, cultural and economic factors on morbidity and mortality, and what many refer to as the structural vulnerabilities that produce poor health as well as poor health care. The biomedical linkage of the 'inner' molecular world and the 'outer' social world may direct the attention of scientists and public health experts to the broader social, political and cultural arrangements that structure the risk of exposure, or it may occlude the social through a narrow and reductionist emphasis on molecular processes alone (Shostak, 2003, p. 2337).

Much of the burden of cancer and other health disparities found in these six cases around the world are results not just of infectious agents, but of structural inequalities that leave the infectious agents as well as early cancer manifestations to persist in certain bodies. While an outward turn is needed in these cases, it is unfortunately largely being supplanted by a biological approach. We therefore caution against a biomedicalization (or pharmaceuticalization) of health, illness and health promotion. While we do not advocate for a return to the stigmatization, discrimination and panics of the early HIV/AIDS epidemic, we believe it is crucial to call attention to how sexuality and gender, and sexual risk factors are co-constituted with other structural inequalities in shaping the epidemiology of disease, including cancers. The ways sexuality and health intertwine in biomedical and cultural rhetoric is significant for health equity. While sexualization can be culturally stigmatizing, as was the case in STI-cancer rhetoric and, especially, in HIV/AIDS, it can also produce recognition of diverse sexual practices and communities and point to sites of needed intervention.

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