26. TÜRK KLİNİK MİKROBİYOLOJİ VE İNFEKSİYON HASTALIKLARI KONGRESİ

Aslında Her Şey Viraldir !

KANSER YAPAN VİRUSLAR

Dr. Zeynep Tekin Taş
Sincan Eğitim ve Araştırma Hastanesi
Enfeksiyon Hastalıkları ve Klinik Mikrobiyoloji

Kanser Yapan Viruslar

ELSEVIER

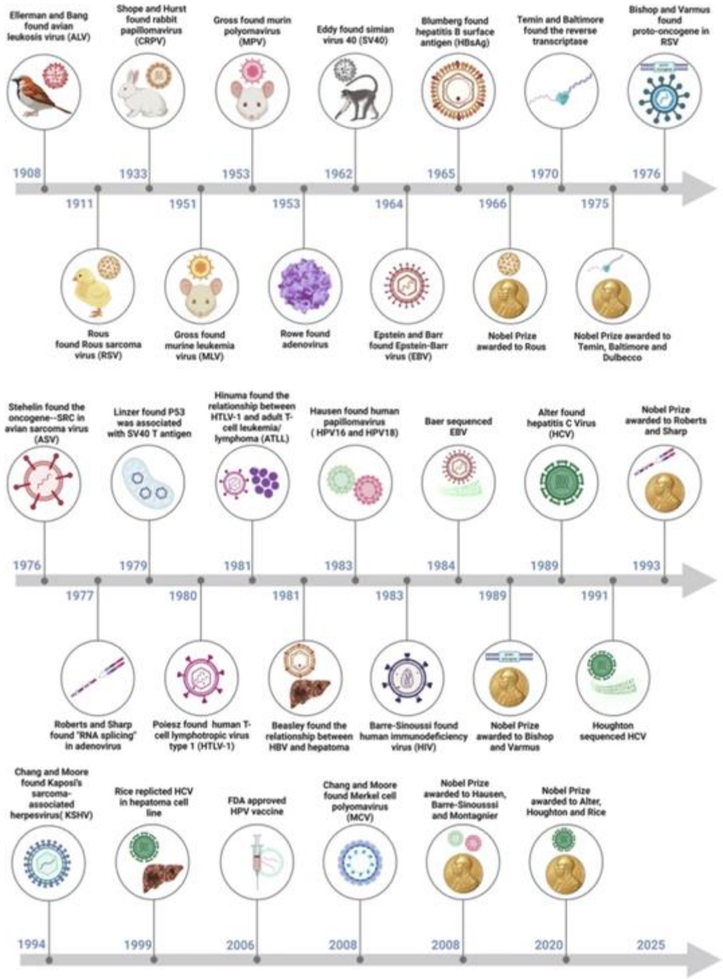
The Lancet

Volume 283, Issue 7335, 28 March 1964, Pages 702-703

Preliminary Communications

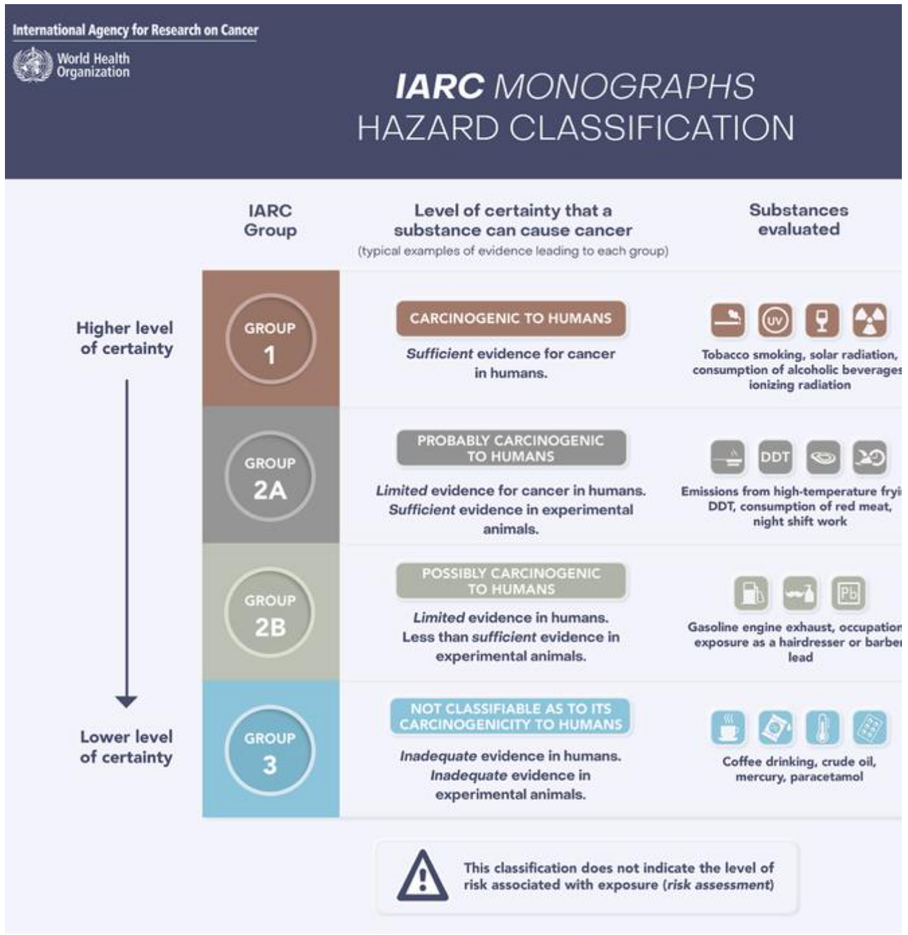
VIRUS PARTICLES IN CULTURED LYMPHOBLASTS FROM BURKITT'S LYMPHOMA

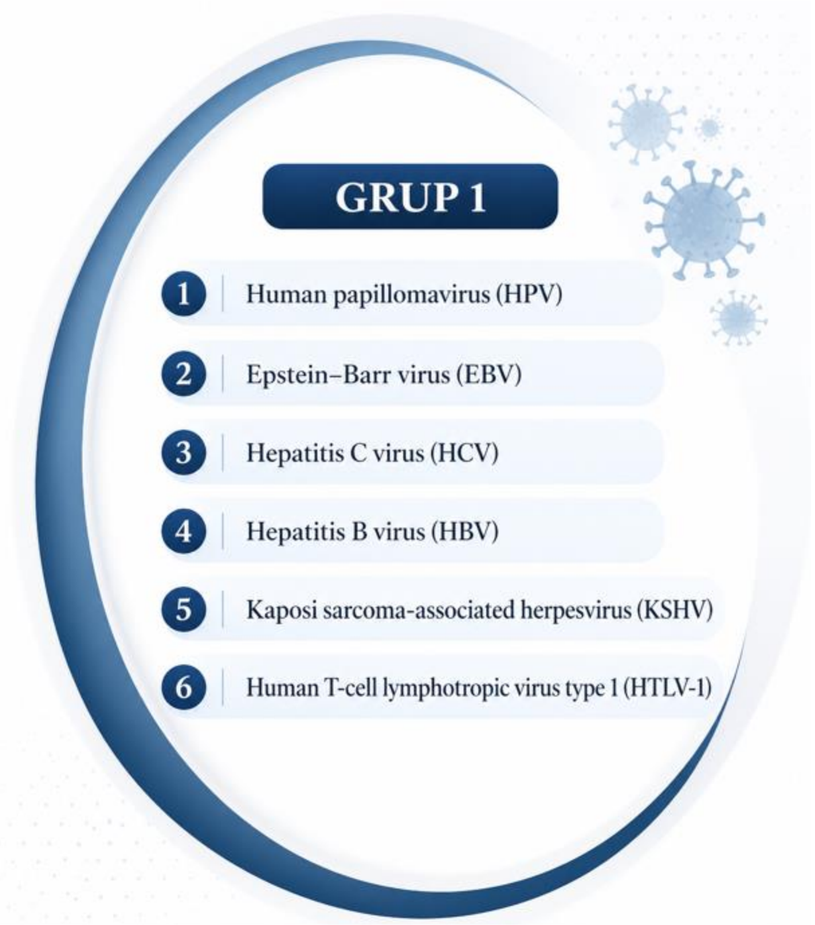
M.A Epstein M.A., M.D. Cantab., D.SC., PH.D. Lond., F.C.PATH., B.G Achong M.B. Dubl., Y.M Barr B.A. Dubl.

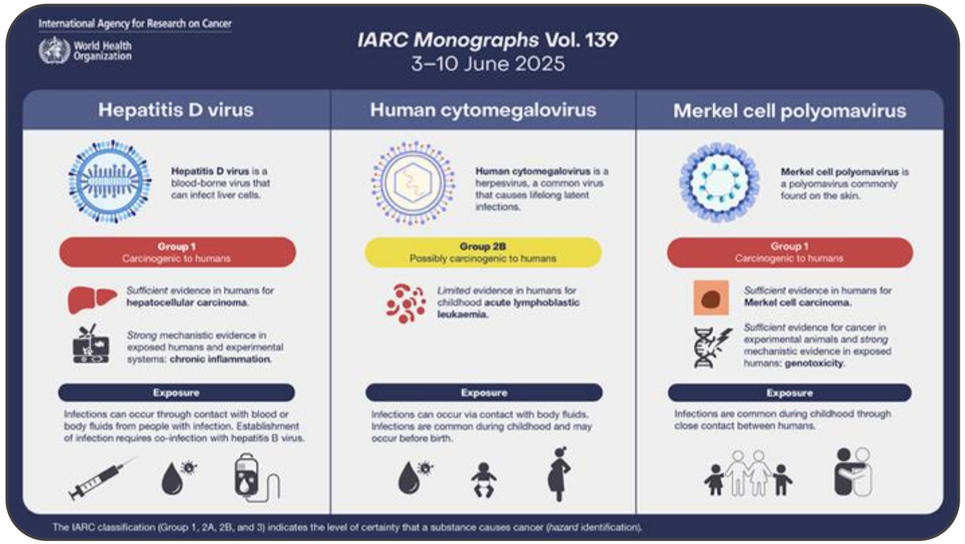


doi:10.1038/s41392-025-02197-9

Kanser Yapan Viruslar







Lancet Oncol. Author manuscript; available in PMC: 2025 Dec 26.

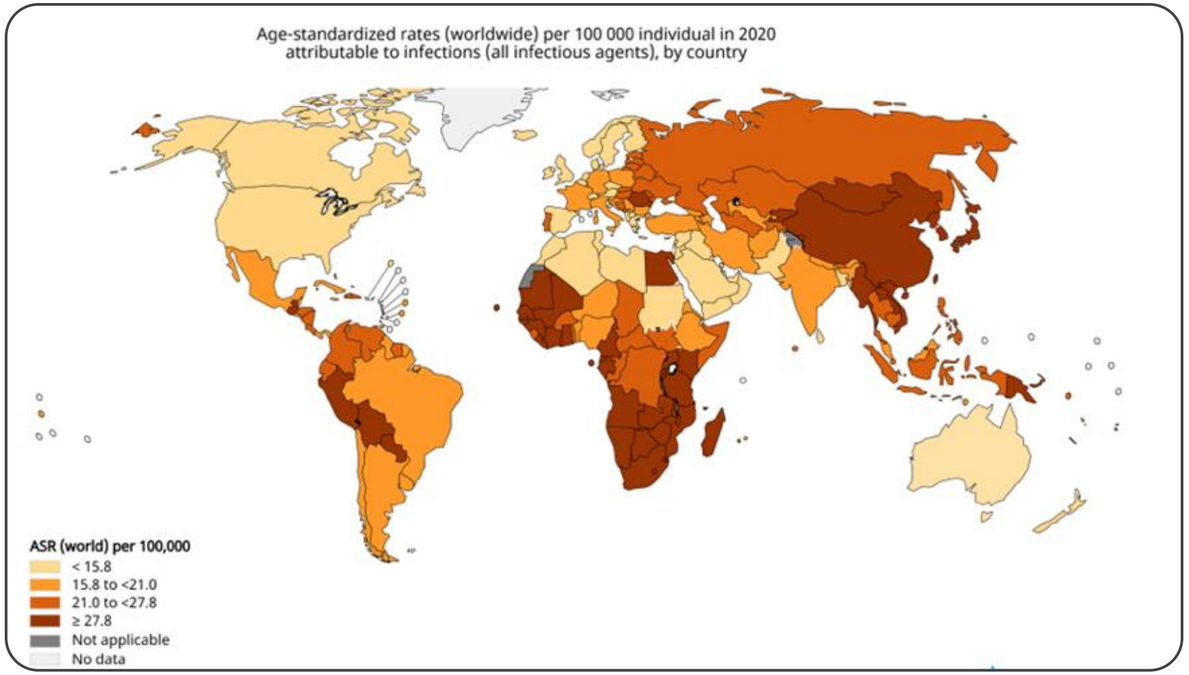
Published before final editing as: Lancet Oncol. 2025 Jun 26:S1470-2045(25)00403-6. doi: 10.1016/S1470-2045(25)00403-6

Carcinogenicity of hepatitis D virus, human cytomegalovirus, and Merkel cell polyomavirus

Margaret R Karagas, John Kaldor, Martin Michaelis, Mazvita M Muchengeti, Dulce Alfaiate, Ilona Argirion, Xiaohua Chen, Celso Cunha, Sébastien Hantz, Virve Koljonen, Hélène C Laude, Weng-Onn Lui, Michael M Nevels, Thomas R O'Brien, Michael E Scheurer, Antoine Touze, Joseph L Wiemels, Lamia Benbrahim-Tallaa, Aline de Conti, Caterina Facchin, Federica Madia, Elisa Pasqual, Roland Wedekind, Inmaculada Aguilera-Buenosvinos, Gary Clifford, Xiaobei Deng, Rachmad A Dongoran, Sarra Ezzemni, Yue Huang, Eero Suonio, Yue Zhai, Heidi Mattock, Mary K Schubauer-Berigan *, Andrew T Kunzmann *

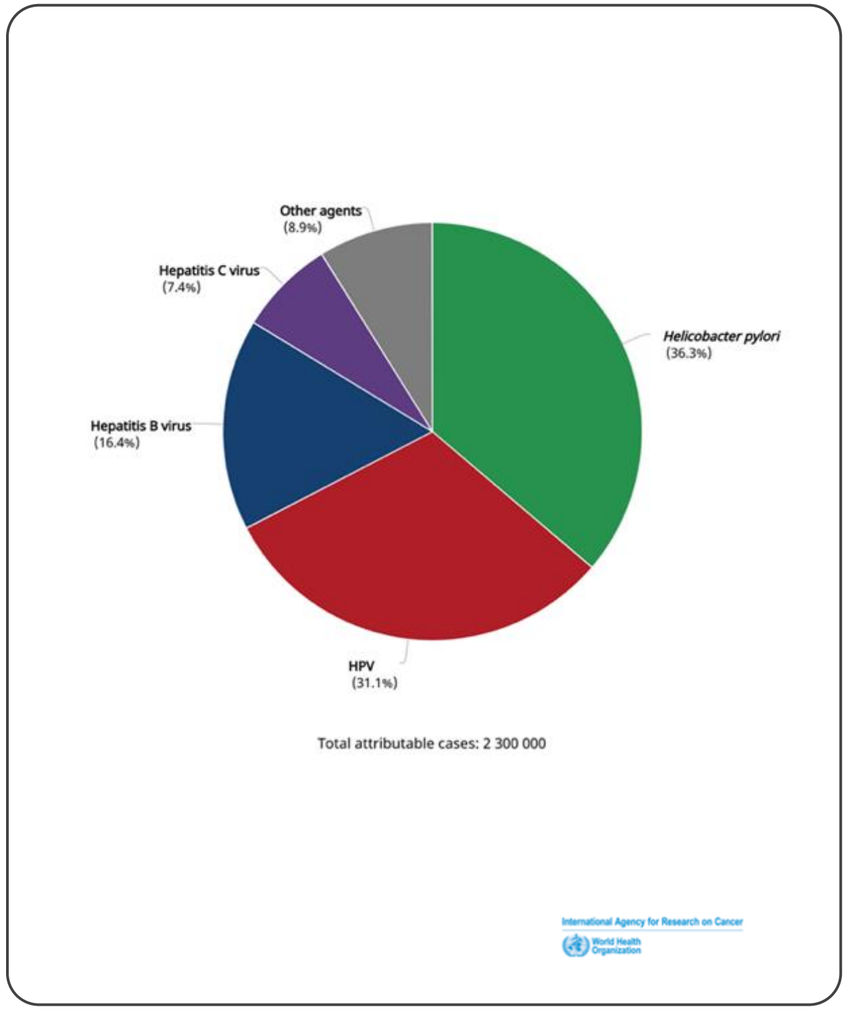
- **Haziran 2025'te IARC, hepatit D virüsü ve Merkel hücreli poliomavirüsü için yeterli kanıt bulunması nedeniyle bu etkenleri Grup 1 olarak sınıflandırdı**
- **Sitomegalovirüsü ise çocukluk çağı akut lenfoblastik lösemi ile ilişkisine dair sınırlı kanıt nedeniyle Grup 2B grubuna aldı**
- **HIV onkojenik virüslerin etkisini artırarak kansere dolaylı katkı sağladığından, IARC ayrıca HIV için ayrı bir kanserojen sınıflandırma yapmamakta**

Aslında Herşey Viraldir !



- **Enfeksiyöz ajanlara atfedilen kanserlerin yarıdan fazlası viral etkenlerle ilişkili**
- **Viral enfeksiyonlar, dünya genelinde her yıl yaklaşık 1,4 milyon yeni kanser vakasına atfediliyor**
- **Tüm insan tümörlerinin yaklaşık %8'i viral kaynaklıdır**

Kanser Yapan Viruslar



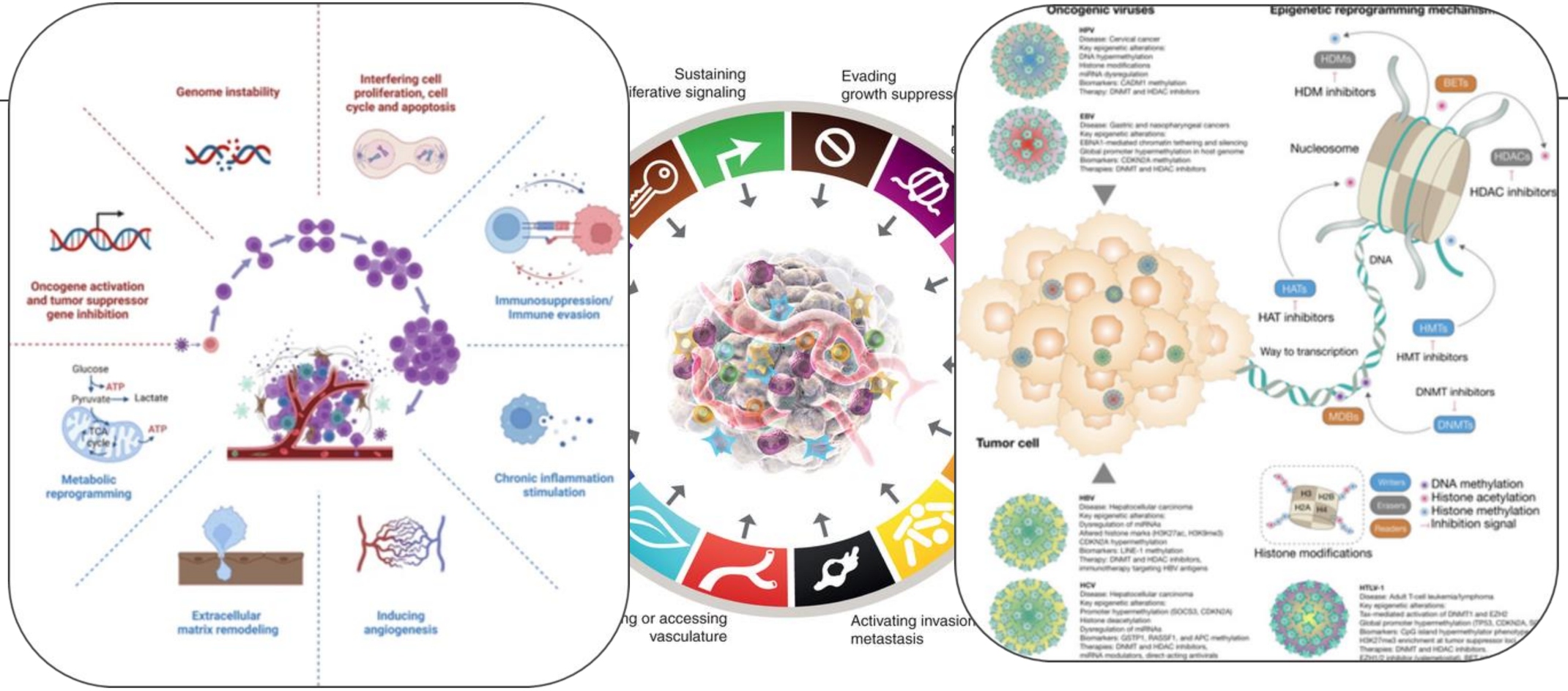
- **Viral kanserlerin %49'u HPV, %26'sı HBV, %11'i HCV, %11'i EBV ve %3'ü diğer virüslerle ilişkilidi**
- **ASIR değerleri belirgin bölgesel farklılıklar gösterirken, düşük önleme ve tedavi olanakları nedeniyle Asya ve Afrika HPV, HBV ve HCV açısından başlıca endemik bölgeler**

Table 1. The age-standardized global incidence and number of cancer cases attributable to viral infections in 2022, by oncoviruses and geographical region

Region	HPV		HBV		HCV		EBV		KSHV		HTLV-1	
	ASIR	NNC	ASIR	NNC	ASIR	NNC	ASIR	NNC	ASIR	NNC	ASIR	NNC
Africa	30.0	137,000	4.6	40,000	2.3	21000	1.1	24,000	2.3	27,000	-	<500
Latin America and the Caribbean	18.5	75,000	2.7	23,000	1.1	9700	0.2	8300	0.4	2800	-	<500
Northern America	14.1	40,000	3.7	26,000	2.6	19000	0.3	5800	0.2	1200	-	<1000
Europe	17.2	99,000	2.8	48,000	1.9	31000	0.3	12,000	0.2	2700	-	<1000
Pacific Ocean	15.4	4400	4.1	2600	1.8	1100	0.4	<500	0.1	<100	-	<100
Asia	16.1	460,000	5.5	330,000	2.5	150000	1.8	120,000	0.1	2500	-	2000
Global	18.5	820,000	3.9	470,000	2.0	230000	1.4	170,000	0.4	36,000	-	4000

Data are ASIR per 100, 000 person-years. The number of cases have been rounded to two significant digits. ASIR, age-standardized incidence rate; NNC, Number of New Cases. "-" represents an incidence too low to calculate. The list of countries for each region or subregion can be found at Cancer today's data & methods section (https://gco.iarc.fr/today)

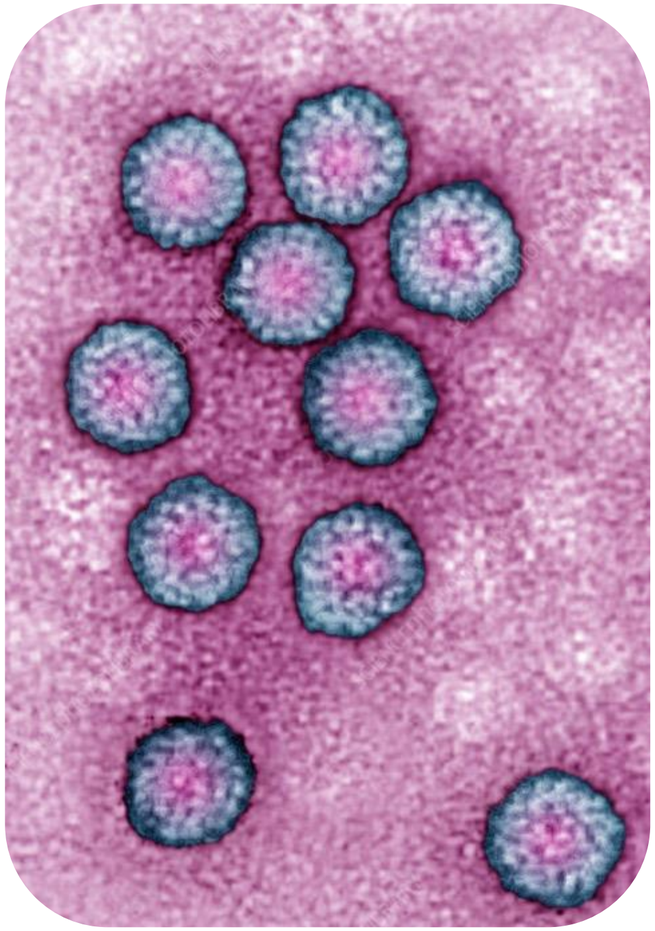
doi:10.1038/s41392-025-02197-9



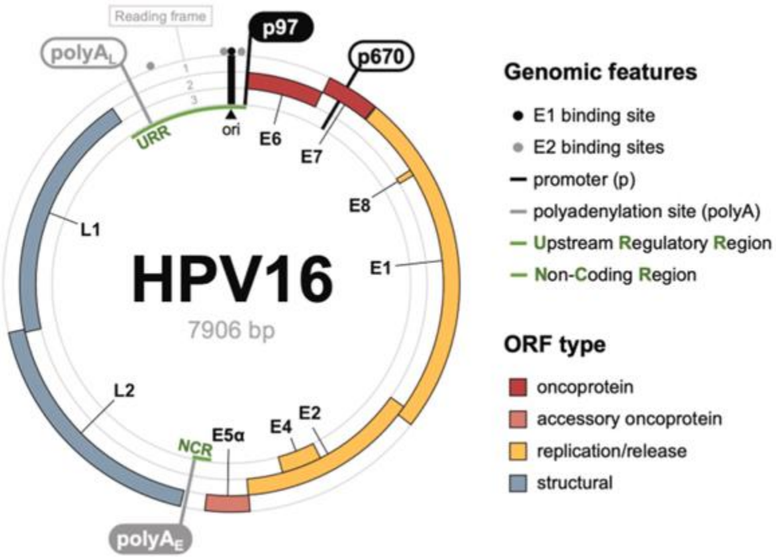
doi: 10.3389/fcimb.2025.1617198

Human Papillomavirus

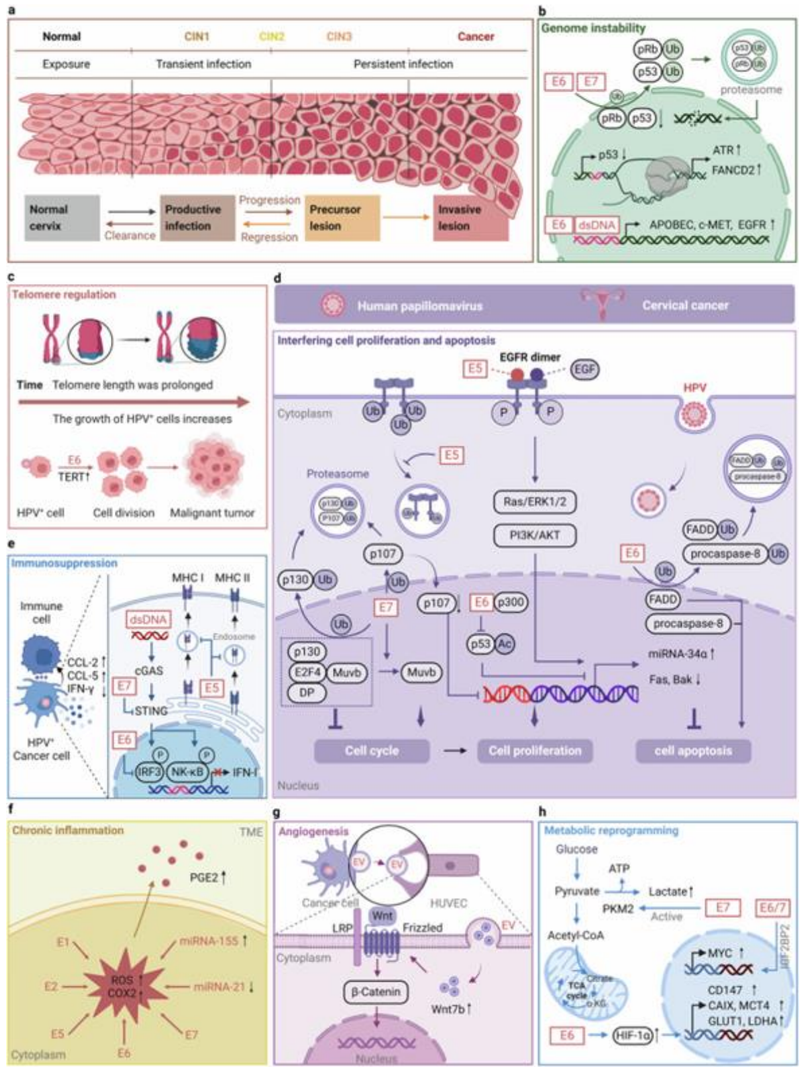
(HPV)



Kanser Yapan Viruslar

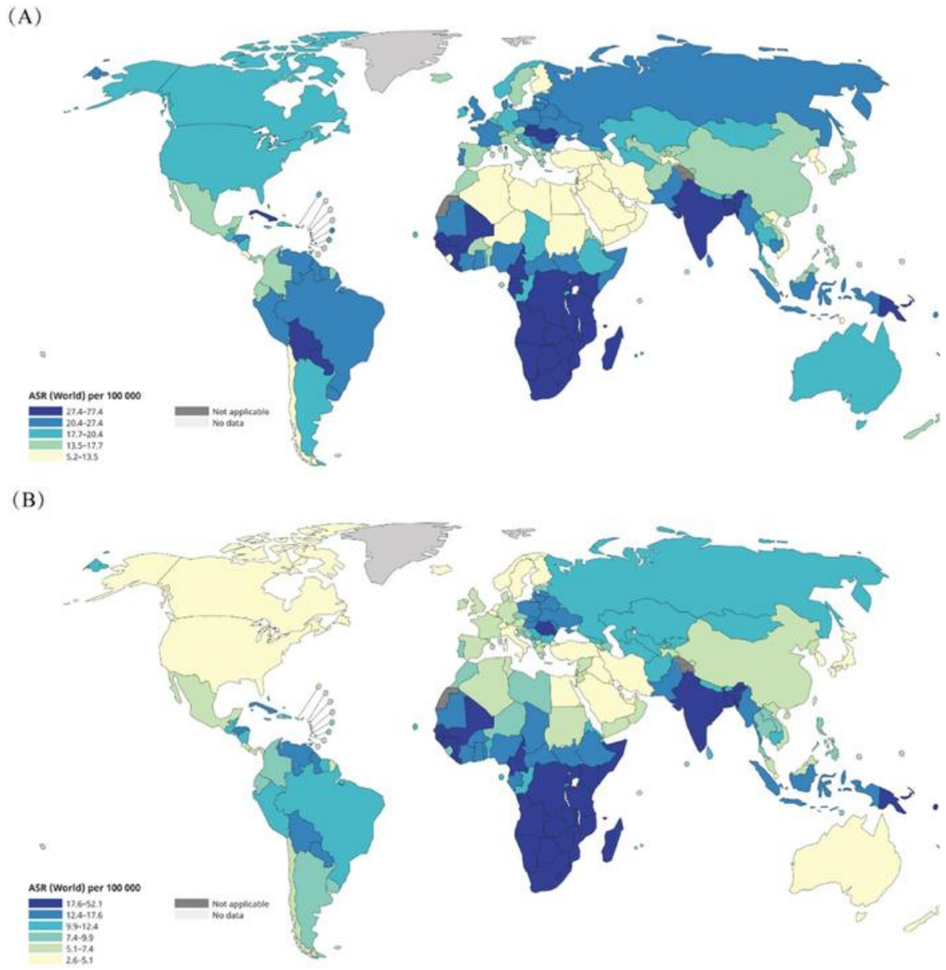


- Kanser HPV için evrimsel avantaj sağlamaz
- Onkogenez, viral süreçlerin yan ürünüdür
- Kanser hücrelerinde viral replikasyon sonlanır



doi:10.1038/s41392-025-02197-9

- Serviks kanseri
- Anal kanser
- Orofarenks kanseri
- Vajinal kanser
- Vulvar kanser
- Penil kanser
- Oral kavite kanseri
- Larenks kanseri



doi:10.1038/s41598-025-06700-8

HPV cancer burden by anatomical site, country, and region in 2022

Jingyuan Zhang [1,#], Yong Ke [2,#], Chi Chen [1,#], Zhihao Jiang [1], Heng Liu [3], Yanhong Liu [4,✉], Hong Cao [1,✉]

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		Total	Men		Women		
Incidence	New cases	New cases attributed to HPV infection	New cases	New cases attributed to HPV infection	New cases	New cases attributed to HPV infection	ASR (World)
Lip, oral cavity	389,846	16,763	268,999	11,567	120,847	5196	4.00
Oropharynx	106,400	39,794	86,339	32,291	20,061	7503	1.10
Anus	54,306	54,306	24,033	24,033	30,273	30,273	0.54
Larynx	189,191	9460	165,794	8290	23,397	1170	1.90
Vulva	47,336	14,674	0	0	47,336	14,674	0.83
Vagina	18,819	14,679	0	0	18,819	14,679	0.36
Cervix uteri	662,301	662,301	0	0	662,301	662,301	14.10
Penis	37,700	19,227	37,700	19,227	0	0	0.79
All cancer types related to HPV infection	1,505,899	831,204	582,865	95,407	923,034	735,796	
Mortality rate				0		0	
Lip, oral cavity	188,438	8103	130,808	5625	57,630	2478	1.90
Oropharynx	52,305	19,562	42,818	16,014	9487	3548	0.53
Anus	22,035	22,035	10,874	10,874	11,161	11,161	0.21
Larynx	103,359	5168	90,384	4519	12,975	649	1.00
Vulva	18,579	5759	0	0	18,579	5759	0.30
Vagina	8240	6427	0	0	8240	6427	0.15
Cervix uteri	348,874	348,874	0	0	348,874	348,874	7.10
Penis	13,738	7006	13,738	7006	0	0	0.28
All cancer types related to HPV infection	755,568	422,935	288,622	44,038	466,946	378,897	

Table 1. Estimated number of cases attributable to HPV infection worldwide in 2022.

- **HPV ilişkili kanserlerin çoğu servikal (%75,6), ardından orofarengeal kanserler**
- **Vakaların %88,6'sı ve ölümlerin %89,6'sı kadınlar oluşturmakta**

Sci Rep. 2025 Jul 1;15:21048. doi: 10.1038/s41598-025-06700-8

HPV cancer burden by anatomical site, country, and region in 2022

Jingyuan Zhang [1,#], Yong Ke [2,#], Chi Chen [1,#], Zhihao Jiang [1], Heng Liu [3], Yanhong Liu [4,✉], Hong Cao [1,✉]

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PMCID: PMC12214776 PMID: 40593150

2045 yılına kadar, sekiz kanser türü için vaka sayısının beş kıtanın tamamında artış göstermesi (%3,9 ile %62,3) beklenmekte

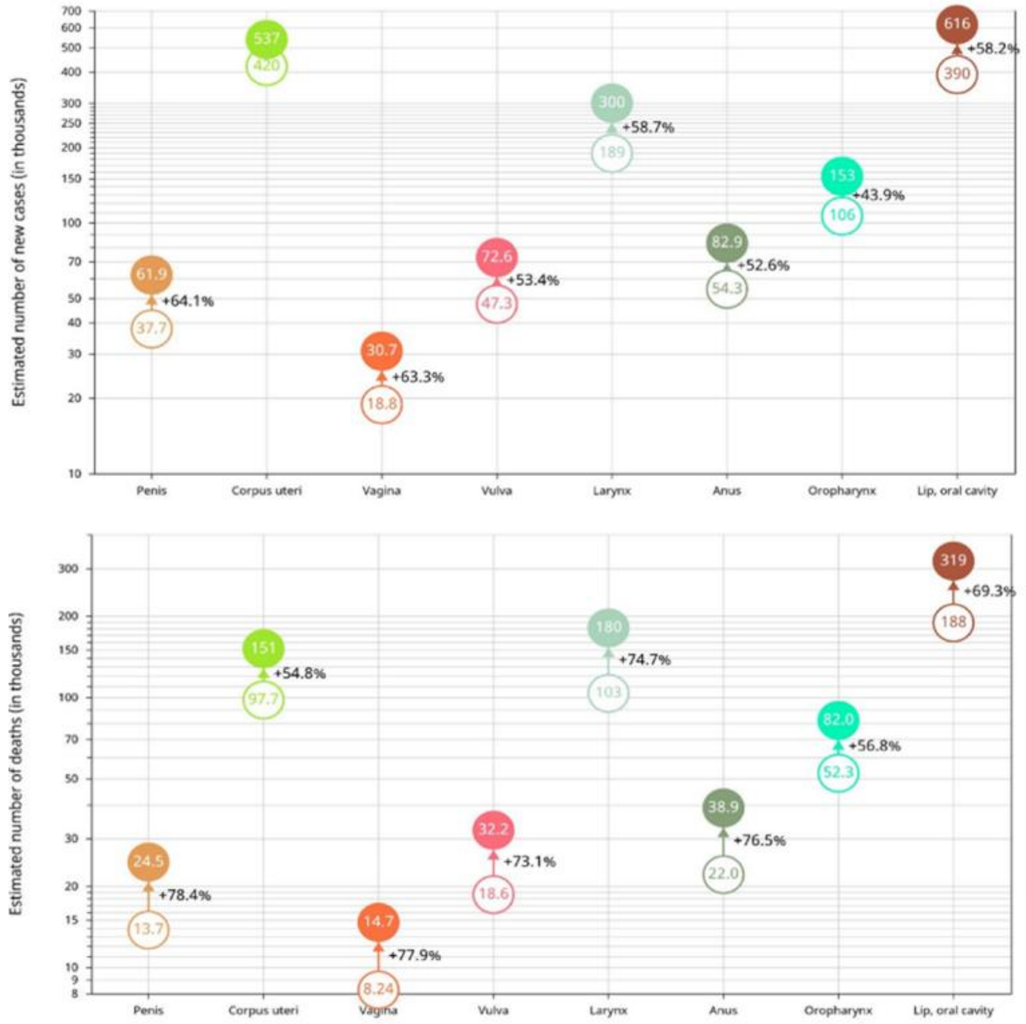
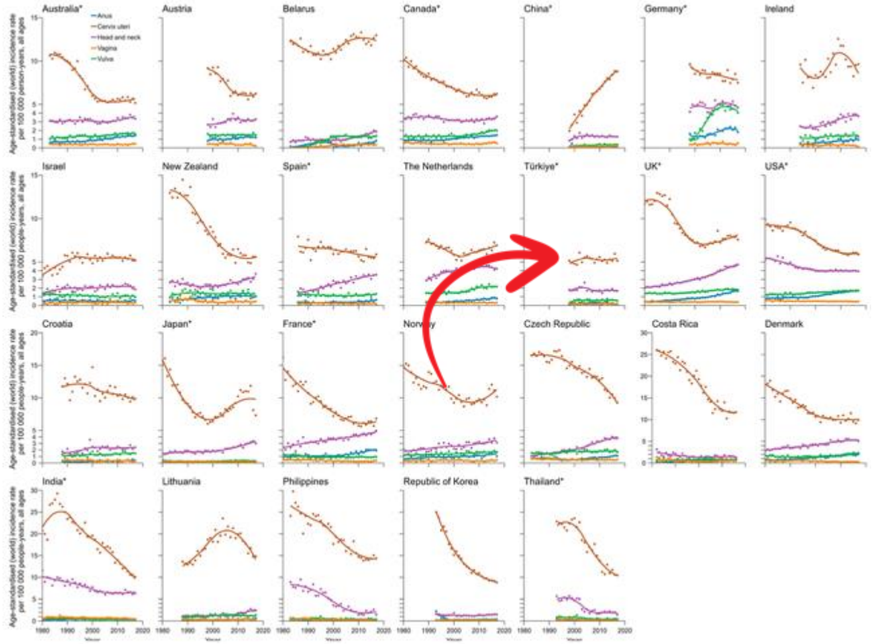


Fig. 3. The absolute incidence and mortality growth trends of eight cancers from 2022 to 2045.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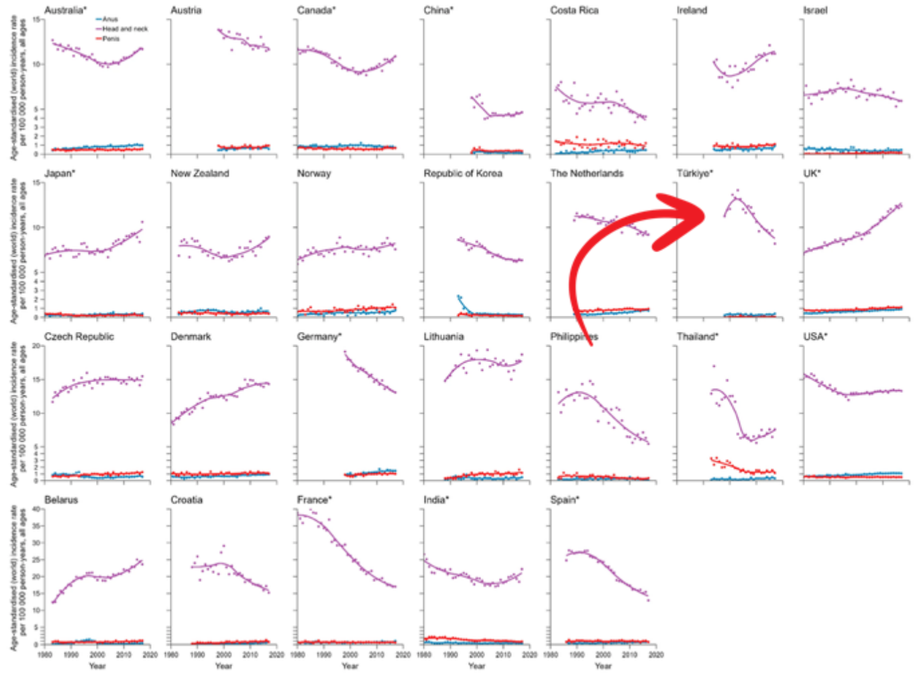
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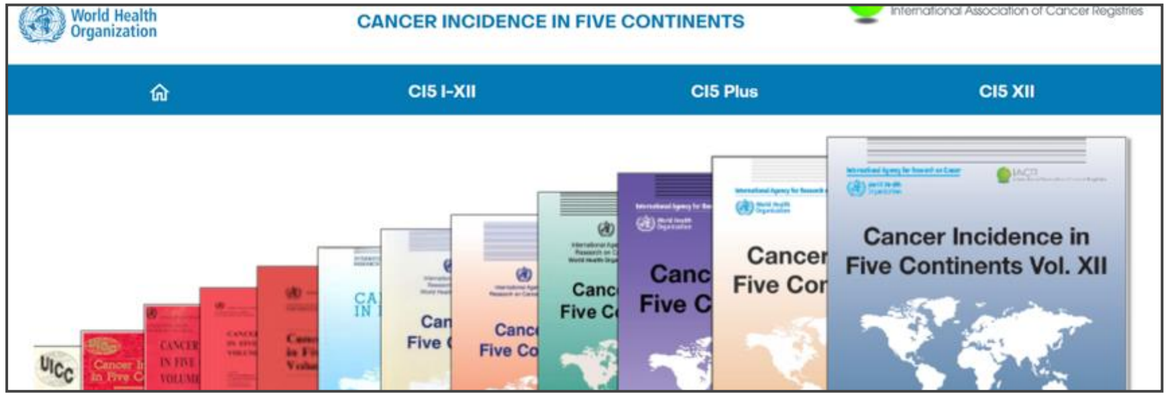
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THE LANCET

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ARTICLES · Volume 404, Issue 10451, P435-444, August 03, 2024 Download Full Issue

Causal attribution of human papillomavirus genotypes to invasive cervical cancer worldwide: a systematic analysis of the global literature

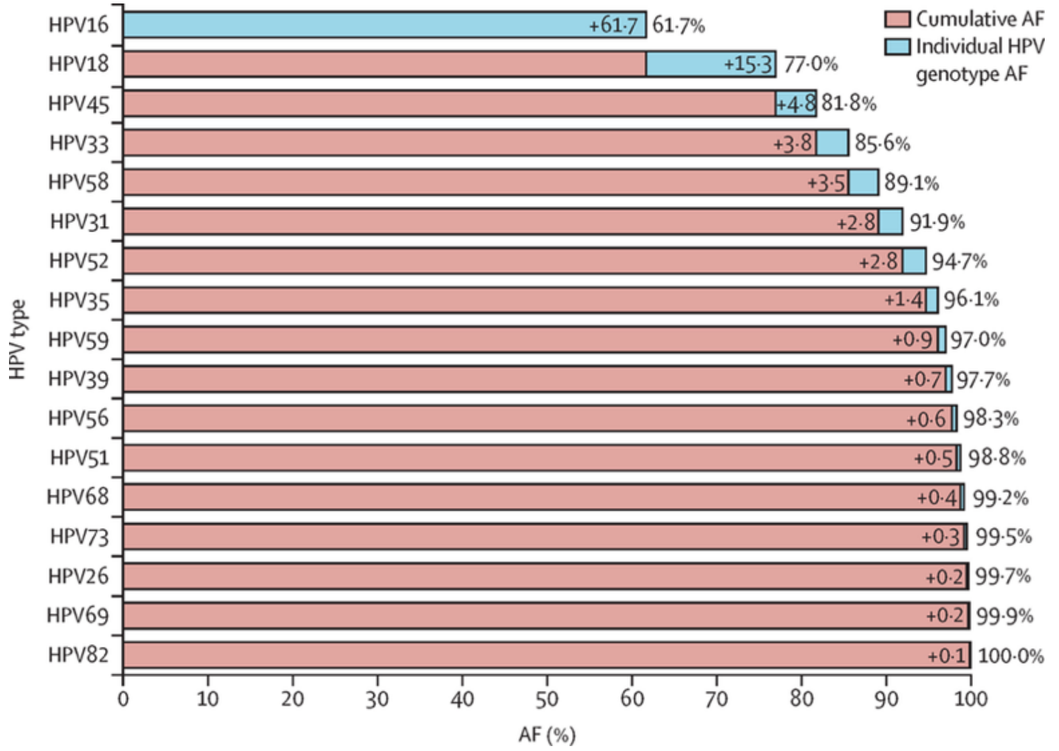
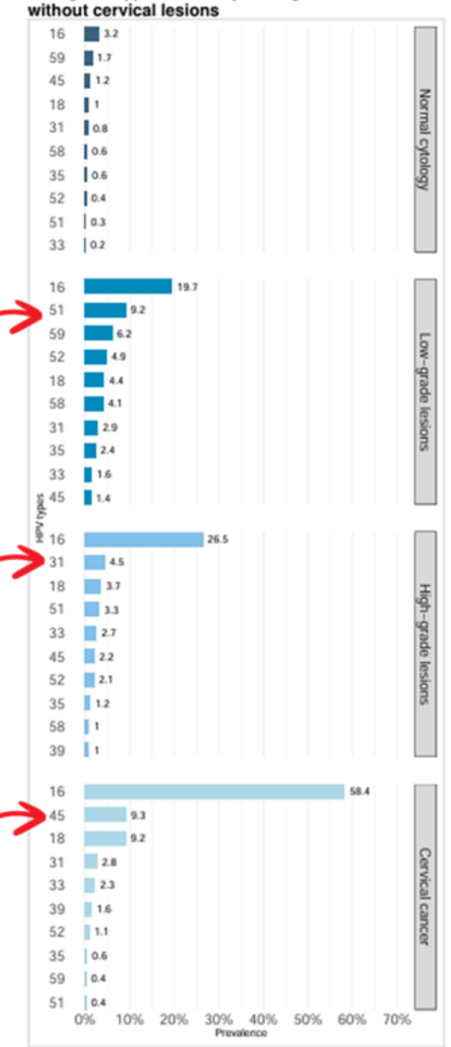


Figure 1. Comparison of the ten most frequent HPV oncogenic types in Turkey among women with and without cervical lesions



ICO/IARC Information Centre on HPV and Cancer

Turkey

Human Papillomavirus and Related Cancers, Fact Sheet 2023 (2023-03-10)

HPV INFORMATION CENTRE

I. Key data on HPV and HPV-related cancers

Table 1. Crude incidence rates of HPV-related cancers

	Male	Female
Cervical cancer	-	5.93
Anal cancer	0.26	0.29
Vulva cancer	-	0.67
Vaginal cancer	-	0.26
Penile cancer	0.06	-
Oropharyngeal cancer	0.41	0.12
Oral cavity cancer	3.38	1.63
Laryngeal cancer	9.14	0.71

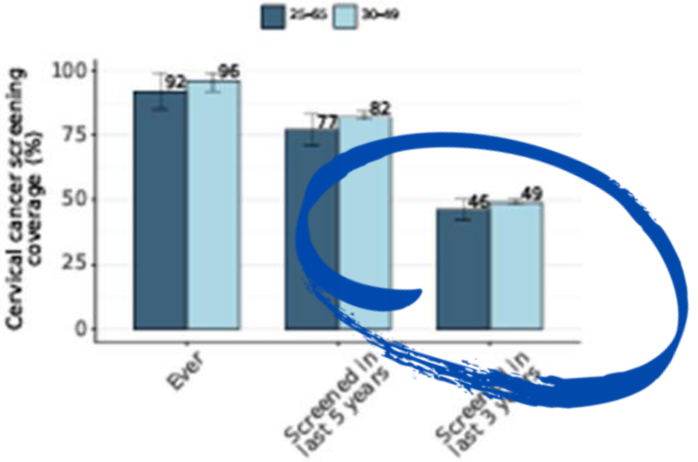
Table 2. Burden of cervical cancer

	Incidence	Mortality
Annual number of new cases/deaths	2532	1245
Crude rate	5.93	2.92
Age-standarized rate	4.81	2.23
Cumulative risk 0-74 years (%)	0.51	0.25
Ranking of cervical cancer (all years)	12th	12th
Ranking of cervical cancer (15-44 years)	5th	8th

Table 3. Burden of cervical HPV infection Turkey

	No. Tested	% (95% CI)
HPV 16/18 prevalence:		
Normal cytology	2478	4.2 (3.5-5.1)
Low-grade cervical lesions	137	24.1 (17.7-31.9)
High-grade cervical lesions	620	30.2 (26.7-33.9)
Cervical cancer	531	67.6 (63.5-71.4)

Figure 2. Estimated coverage of cervical cancer screening in Turkey*



AŞI ÇAĞINDA HPV GENOTİPLERİ

Journal of the National Cancer Center 5 (2025) 586–592

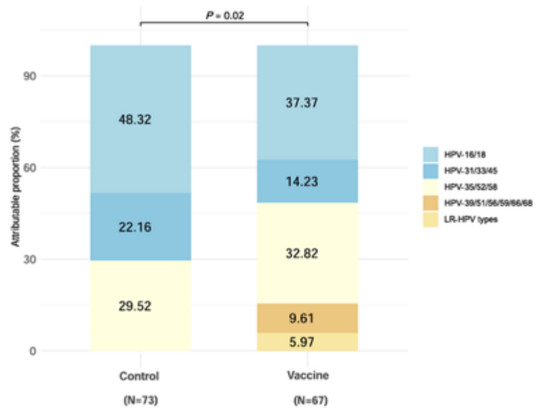
Contents lists available at ScienceDirect

Journal of the National Cancer Center

journal homepage: www.elsevier.com/locate/jncc

Full Length Article

Ten-year evaluation of HPV vaccine influence on non-vaccine-type-related cervical infections and precancers in Chinese females: follow-up from a randomized control trial



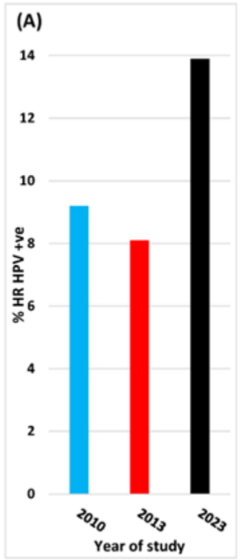
viruses

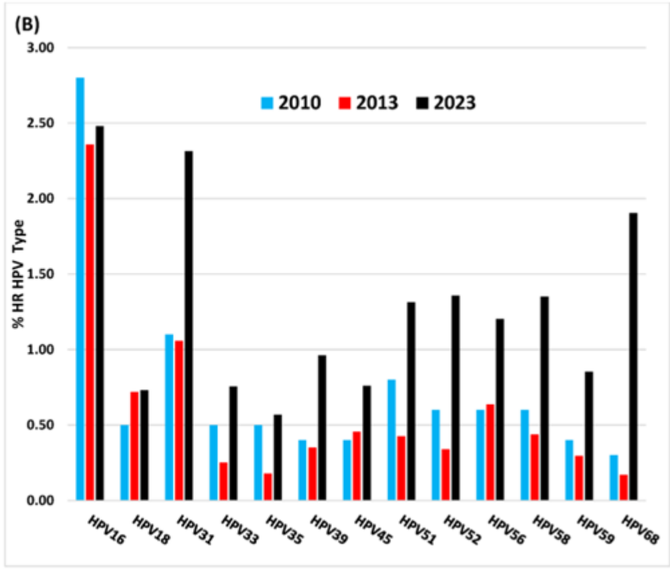
MDPI

Perspective

Update on Effects of the Prophylactic HPV Vaccines on HPV Type Prevalence and Cervical Pathology

Ian N. Hampson [1,*] and Anthony W. Oliver [2]





AŞI ÇAĞINDA HPV GENOTİPLERİ

The Journal of Infectious Diseases

MAJOR ARTICLE

Human Papillomavirus Genotype Replacement: Still Too Early to Tell?

Irene Man,[1,2] Simopekka Vänskä,[3,4] Matti Lehtinen,[5,6] and Johannes A. Bogaards[1,7]

[1]Centre for Infectious Disease Control, National Institute for Public Health and the Environment, Bilthoven, Netherlands, [2]Department of Biomedical Data Sciences, Leiden University Medical Center, Leiden, Netherlands, [3]Infectious Disease Control and Vaccinations, National Institute for Health and Welfare, Helsinki, Finland, [4]School of Health Sciences, University of Tampere, Finland, [5]Department of Laboratory Medicine, Karolinska Institute, Stockholm, Sweden, [6]Division of Infections and Cancer Epidemiology, Deutsches Krebsforschungszentrum, Heidelberg, Germany, [7]Department of Epidemiology and Biostatistics, Amsterdam University Medical Center, Vrije Universiteit Amsterdam, Amsterdam, Netherlands

Conclusions

Although postvaccination surveillance thus far is reassuring, it is still too early to preclude type replacement. Monitoring of NVTs remains pivotal in gauging population-level impacts of HPV vaccination.

PLOS Pathogens

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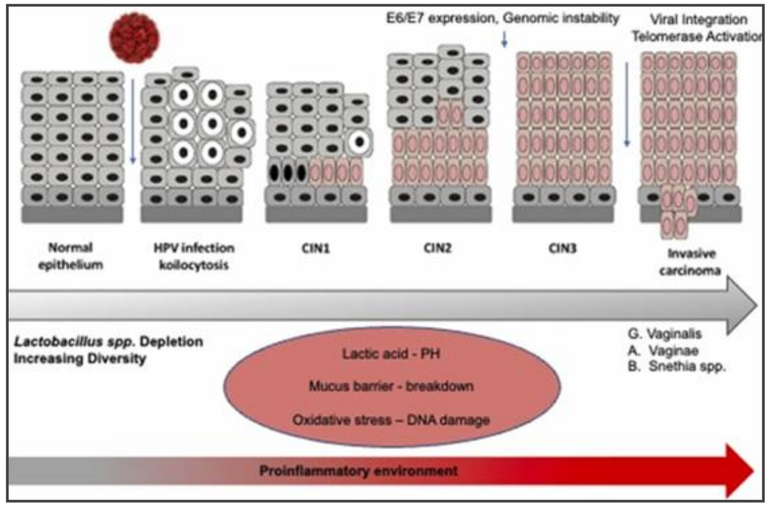
RESEARCH ARTICLE

Cervicovaginal microbiome and natural history of HPV in a longitudinal study

Mykhaylo Usyk, Christine P. Zolnik, Philip E. Castle, Carolina Porras, Rolando Herrero, Ana Gradissimo, Paula Gonzalez, Mahboobeh Safaeian, Mark Schiffman, Robert D. Burk, Costa Rica HPV Vaccine Trial (CVT) Group

Published: March 26, 2020 • https://doi.org/10.1371/journal.ppat.1008376



Review 6 August 2020

High-Risk Human Papillomavirus and Tobacco Smoke Interactions in Epithelial Carcinogenesis

Francisco Aguayo[1,2,*], Juan P. Muñoz[3], Francisco Perez-Dominguez[4], Diego Carrillo-Beltrán[4], Carolina Oliva[4], Gloria M. Calaf[3,5], Rances Blanco[4] and Daniela Nuñez-Acurio[4]

1 Universidad de Tarapacá, Arica 1000000, Chile
2 Advanced Center for Chronic Diseases (ACCDiS), Facultad de Medicina, Universidad de Chile, Santiago 8330024, Chile
3 Instituto de Alta Investigación, Universidad de Tarapacá, Arica 1000000, Chile
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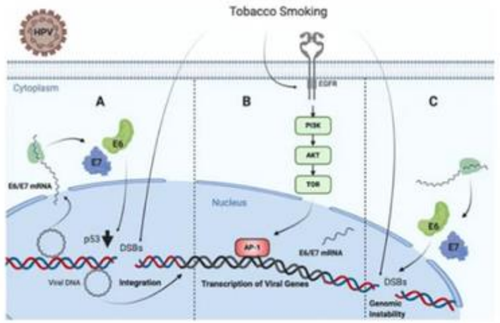


Figure 1. Tobacco smoke cooperates with high-risk human papillomavirus (HR-HPV) for increased DNA damage in epithelial cells. (**A**) Tobacco smoke causes E6/E7 oveerexpression and DNA damage in cells harboring HPV episomal forms, inducing p53 downregulation and potentially promoting viral genome integration [101]. (**B**) Tobacco smoke promotes EGFR/PI3K/AKT activation inducing AP-1 recruitment to the LCR and activating the HR-HPV early promoter, thus increasing E6/E7 expression [103]. (**C**) Both tobacco smoke and E6/E7 oncoproteins cooperate for increasing genomic instability in infected cells [101,138].

The effectiveness of HPV vaccination against invasive cervical cancer and related precancerous lesions: a multinational target trial emulation study

Marta Alcalde-Herraiz, Mike Du*, Aina Sanchez-Parada, Talita Duarte-Salles, Anna Palomar-Cros, Agustina Giuliodori, Antonella Delmestri, Hedvig Marie Egeland Nordeng, Nhung T H Trinh, Saeed Hayati, Daniel Prieto-Alhambra, Marti Català†, Albert Prats-Uribe†*

Summary

Background Human Papillomavirus (HPV) vaccines prevent HPV infection and related disease. 15 years after the first HPV vaccination programmes were launched in Europe, their long-term effectiveness can now start to be assessed. We designed a target trial emulation study to estimate the effectiveness of HPV vaccination in preventing invasive cervical cancer and high-grade precancerous lesions using three primary care databases.

Lancet Prim Care 2026; 2: 100114

Published Online March 16, 2026

https://doi.org/10.1016/

- **81.863 aşılanmış ve 46.357 aşılanmamış kadın**
- **Her kohortta beşten az servikal kanser vakası**
- **15 yıl sonraki meta-analitik aşı etkinliği,**

  CIN2+'ya karşı %42, konizasyona karşı %58

HERD EFFECT

Herd effect of human papillomavirus vaccination on incidence of high-grade cervical lesions: a population-based cohort study in Sweden

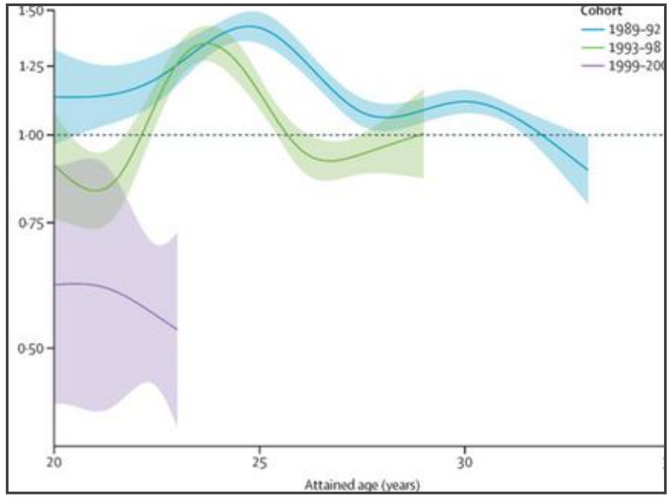
Eva Meglic, Alexander Ploner, Mark Clements, Miriam Elfström, Jiayao Lei

Summary

Background Human papillomavirus (HPV) vaccination has substantially reduced the incidence of high-grade cervical lesions (HSIL+) among vaccinated individuals. However, indirect effects on unvaccinated populations remain unclear. We assessed herd effects by examining age-varying HSIL+ incidence among unvaccinated women in Sweden.

Lancet Public Health 2026; 11: e35–43

See Comment page e4



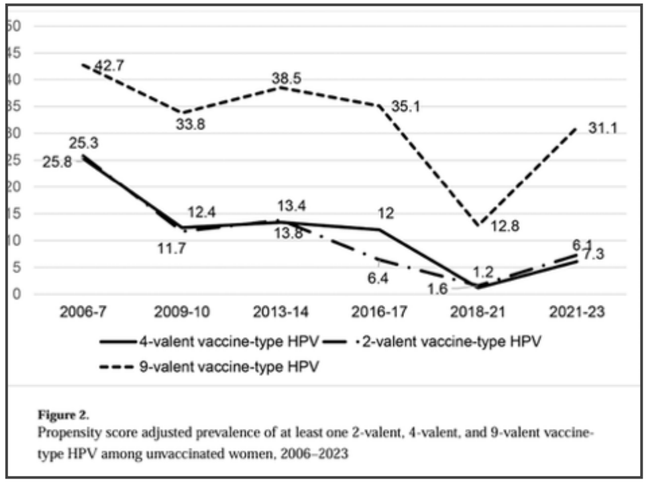


Figure 2.
Propensity score adjusted prevalence of at least one 2-valent, 4-valent, and 9-valent vaccine-type HPV among unvaccinated women, 2006–2023

Published in final edited form as:

JAMA Pediatr. 2025 December 01; 179(12): 1326–1334. doi:10.1001/jamapediatrics.2025.3568.

Real-World Effectiveness and Herd Protection 17 Years After Human Papillomavirus Vaccine Introduction

India introduces human papillomavirus vaccination

On Feb 28, 2026, Indian Prime Minister Narendra Modi unveiled a national campaign to promote human papillomavirus (HPV) vaccination to prevent cervical cancer. During the 90-day drive, 11·5 million girls aged 14 years will be administered the vaccine for free at government-run health facilities across the country. "This is the largest free HPV vaccination campaign in history", said WHO Director-General Tedros Adhanom Ghebreyesus (WHO, Geneva, Switzerland). After the current campaign ends, the Indian health ministry will integrate the HPV vaccine into the Universal Immunisation Programme (UIP) and make it available at government health facilities during routine immunisation days, and cost-effectiveness of the vaccine, the National Technical Advisory Group on Immunization in June, 2022, recommended inclusion of the HPV vaccine in the UIP with a one-time catch-up dose for girls aged 9–14 years, followed by its inclusion in the routine immunisation schedule.

In the present campaign, the single-dose Gardasil-4 vaccine procured through Gavi, the Vaccine Alliance, is being administered. In future, Cervavac, an Indian vaccine originally licensed for two-dose administration, might be used if it is found effective for single-dose administration in an ongoing study that ends in 2027.

"For now, Gardasil-4 is seen as the only viable, immediate solution for HPV vaccination in India, with said: "Proper positioning of the vaccine in the public mind is critical. HPV being sexually transmitted, people conflate it with promiscuity. Communication should centre around how chronic infection, while being asymptomatic, sometimes results in cancer and how we can now prevent it with a vaccine."

"Countering misinformation and overcoming hesitancy by underscoring the long-term benefits for the youth of the country is a key component of our campaign", Nadda said. "The vaccination programme is entirely voluntary, and parental consent is a must to align with the community value system and family autonomy", he pointed out.

The vaccination drive forms a part of India's strategy aimed at

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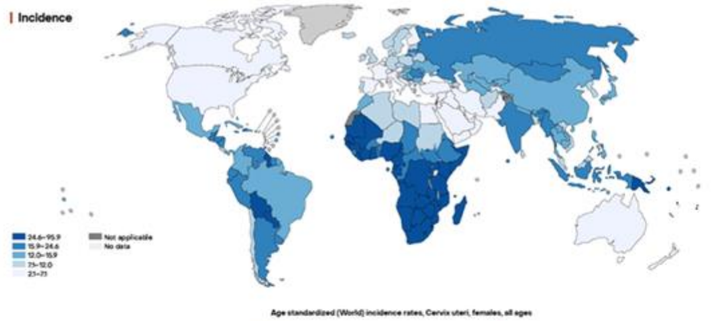
This online publication has been corrected. The corrected version first appeared at thelancet.com/oncology on March 30, 2026

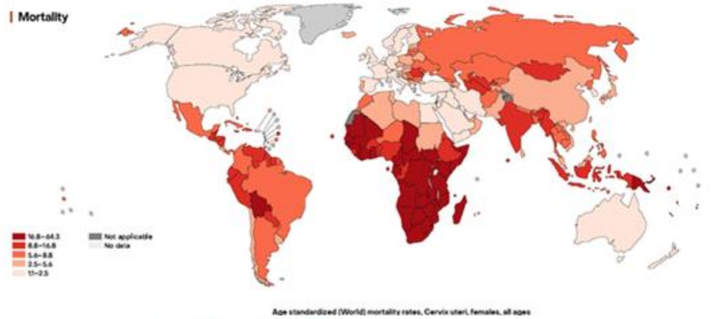
For more on **India's national campaign to promote HPV vaccination** see https://www.pib.gov.in/PressReleasePage.aspx?PRID=2234009&reg=3&lang=2

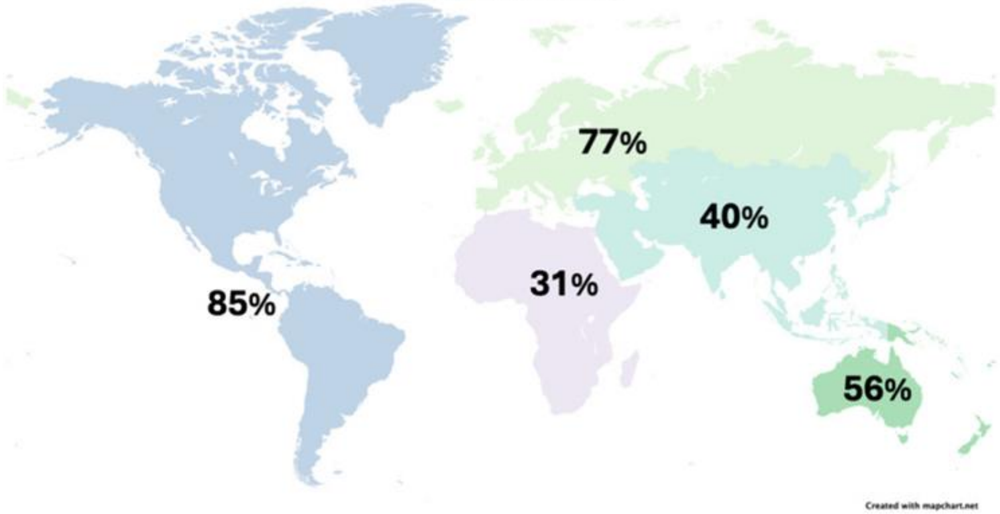
For more on the **parliamentary investigation** see https://hsrii.org/wp-content/uploads/2014/07/72.pdf

For more on **HPV vaccine introduction in Sikkim state** see *Vaccine* 2022; **40** (suppl 1): A17–25

Kanser Yapan Viruslar

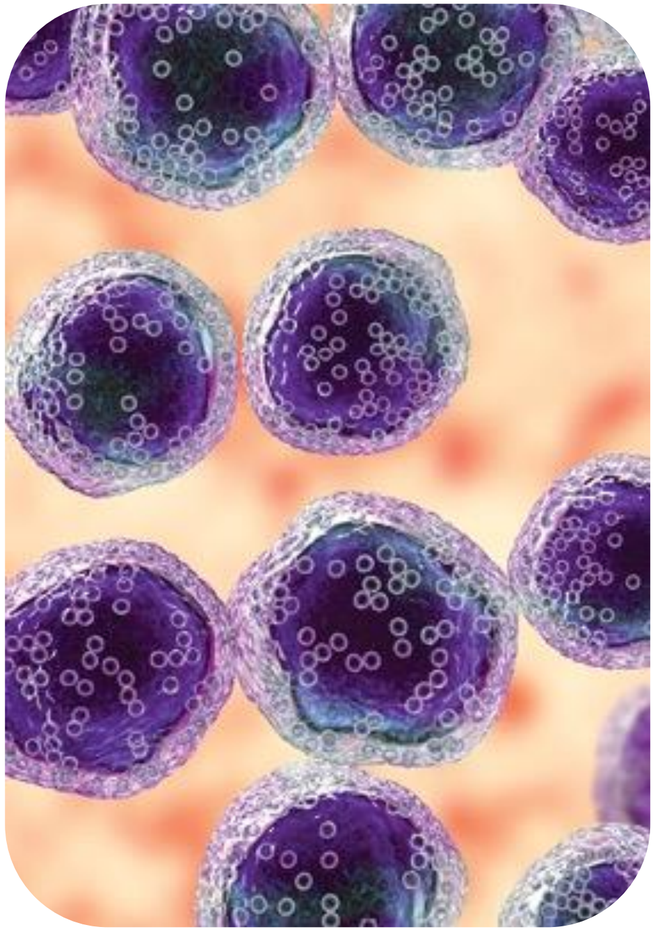






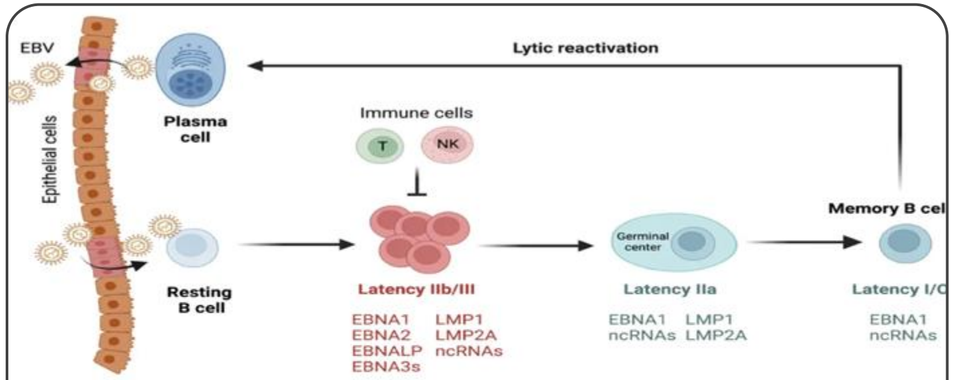
Epstein-Barr Virus

EBV



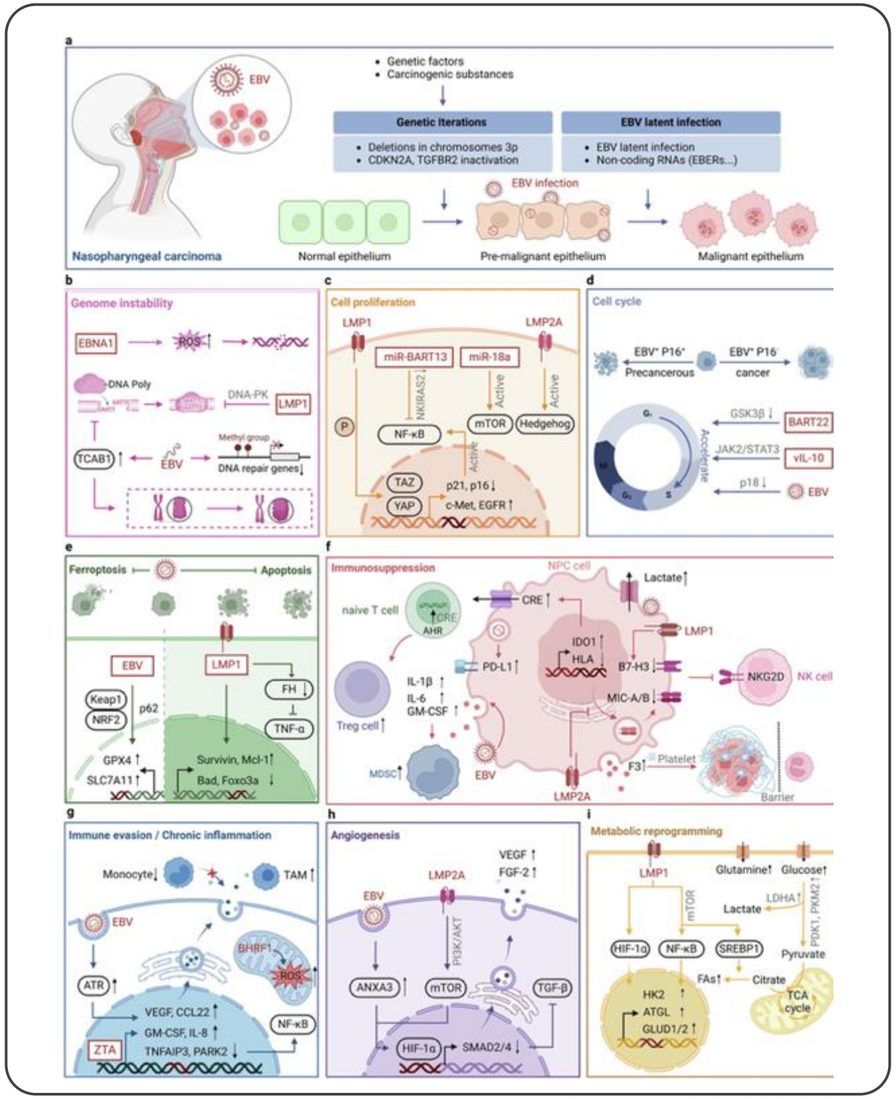
Aslında Herşey Viraldir !

Kanser Yapan Viruslar

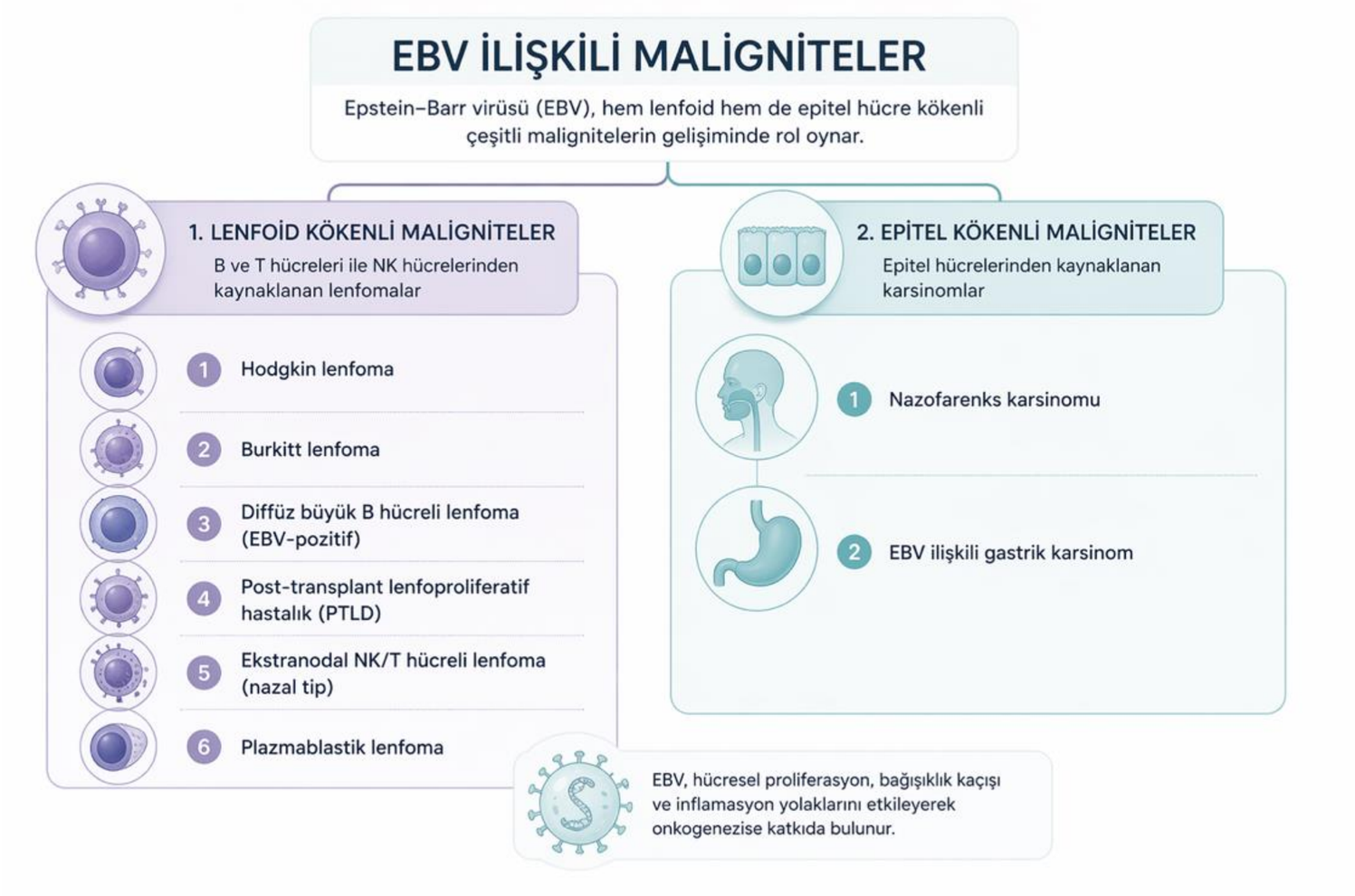


Latency program	Associated malignancies
Latency I	Burkitt lymphoma Gastric cancer*
Latency II	Hodgkin lymphoma Nasopharyngeal carcinoma T/NK cell lymphoma Post-transplant lymphoproliferative disorders**
Latency III	Diffuse large B cell lymphoma

- B hücrelerinde episomal olarak ömür boyu latent kalır
- Tümör gelişimi esas olarak latent fazdaki sınırlı gen ekspresyonuna bağlı
- Latentlik tipleri(III, II, I/0) her biri farklı EBV ilişkili malignitelerle bağlantılıdır
- Düşük gen ekspresyonuyla immün sistemden saklanır



doi:10.1038/s41392-025-02197-9


EBV İLİŞKİLİ MALİGNİTELER
Epstein–Barr virüsü (EBV), hem lenfoid hem de epitel hücre kökenli çeşitli malignitelerin gelişiminde rol oynar.
1. LENFOİD KÖKENLİ MALİGNİTELER
B ve T hücreleri ile NK hücrelerinden kaynaklanan lenfomalar
1 Hodgkin lenfoma
2 Burkitt lenfoma
3 Diffüz büyük B hücreli lenfoma (EBV-pozitif)
4 Post-transplant lenfoproliferatif hastalık (PTLD)
5 Ekstranodal NK/T hücreli lenfoma (nazal tip)
6 Plazmablastik lenfoma
2. EPİTEL KÖKENLİ MALİGNİTELER
Epitel hücrelerinden kaynaklanan karsinomlar
1 Nazofarenks karsinomu
2 EBV ilişkili gastrik karsinom
EBV, hücresel proliferasyon, bağışıklık kaçışı ve inflamasyon yolaklarını etkileyerek onkogenezise katkıda bulunur.

Kanser Yapan Viruslar

International Agency for Research on Cancer
World Health Organization

PRESS RELEASE No. 369

8 July 2025

New study reveals that Epstein–Barr virus infection may increase risk of a broad spectrum of cancer types

Lyon, France, 8 July 2025 – A new study from the International Agency for Research on Cancer (IARC) and its partners explores the link between levels of Epstein–Barr virus (EBV) capsid antigen (VCA-IgA) antibodies, which are produced in response to EBV infection, and cancer risk.

- **VCA-IgA seropozitifliği, tüm kanserler için 4,88 kat artmış risk ile ilişkili**
- **Yüksek antikor düzeyi ile risk artışı ilişkili**
- **Popülasyona atfedilebilir risk: %7,8**

nature communications

Article

https://doi.org/10.1038/s41467-025-60999-5

Epstein Barr virus antibody and cancer risk in two prospective cohorts in Southern China

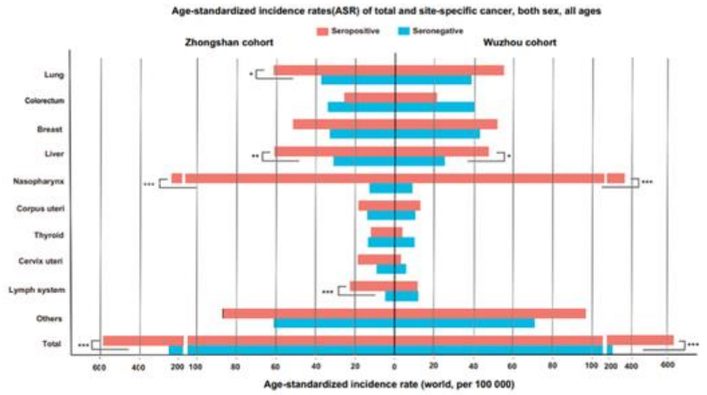
Received: 2 August 2024

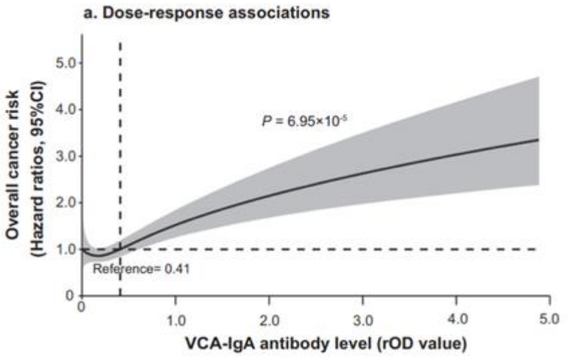
Accepted: 6 June 2025

Published online: 01 July 2025

Check for updates

Ming-Fang Ji[1,14], Yong-Qiao He[2,14] ✉, Min-Zhong Tang[3,4,14], Wen-Qiong Xue[2,14], Xia Yu[1,14], Hua Diao[5], Da-Wei Yang[5], Zhi-Ming Mai[6,7], Io Hong Cheong[8], Zhi-Yang Zhao[5], Biao-Hua Wu[1], Fu-Gui Li[1], Ji-Yun Zhan[9], Chang-Ling Huang[9], Hao-Lin Ma[10], Jun Li[3,4], Yan-Cheng Li[3,4], Tong-Min Wang[2], Ying Liao[2], Xue-Yin Chen[2], Zhi-Heng Liang[1], Shi-Feng Lian[1], Yun Du[11], Xue-Jun Liang[9], Zisis Kozlakidis[12], Jun Ma[2,13] & Wei-Hua Jia[2,5] ✉





Journal of Cancer Research and Clinical Oncology (2022) 148:31–46
https://doi.org/10.1007/s00432-021-03824-y

REVIEW – CANCER RESEARCH

Estimating the global burden of Epstein–Barr virus-related cancers

Yide Wong[1,2,3] · Michael T. Meehan[4] · Scott R. Burrows[5,6] · Denise L. Doolan[1,2,3] · John J. Miles[1,2,3]

Received: 5 April 2021 / Accepted: 28 September 2021 / Published online: 27 October 2021

Cancer type	% estimated global EBV-related case proportion	Estimated incidence range of EBV-related cases	Estimated mortality range of EBV-related cases
NPC	84.6[1]	105,500–120,600	61,600–74,300
GC	7.7–10.4[2]	82,800–116,400	58,200–82,300
HL	45.8–58.3[2]	34,300–52,400	9400–17,400
BL	55[1]	6600[1]	3000–3200
DLBCL	3.6–12.8[2]	4900–27,000	2500–13,300
ENKTL-NT	100[3]	5500–34,700	3000–18,100
Cancer types combined	1.3–1.9[2]	239,700–357,900	137,900–208,700

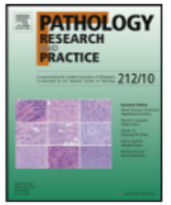
- **EBV, küresel kanser yükünün ~%1,3–1,9'undan ve viral kanserlerin ~%11'inden sorumlu**
- **Nazofarenks kanserlerinin ~%85'i EBV ilişkili, Doğu Asyada sık**

Pathology – Research and Practice 212 (2016) 1021–1026

Contents lists available at ScienceDirect

Pathology – Research and Practice

journal homepage: www.elsevier.com/locate/prp



Original article

Retrospective analysis of oncogenic human papilloma virus and Epstein-Barr virus prevalence in Turkish nasopharyngeal cancer patients☆

- 82 NPC vakası
- %87 EBV pozitifliği
- EBV'nin Türkiye'de nazofaringeal karsinom (NPC) ile yakından ilişkili

Aslında Herşey Viraldir !

Kanser Yapan Viruslar

Journal of Clinical Oncology®
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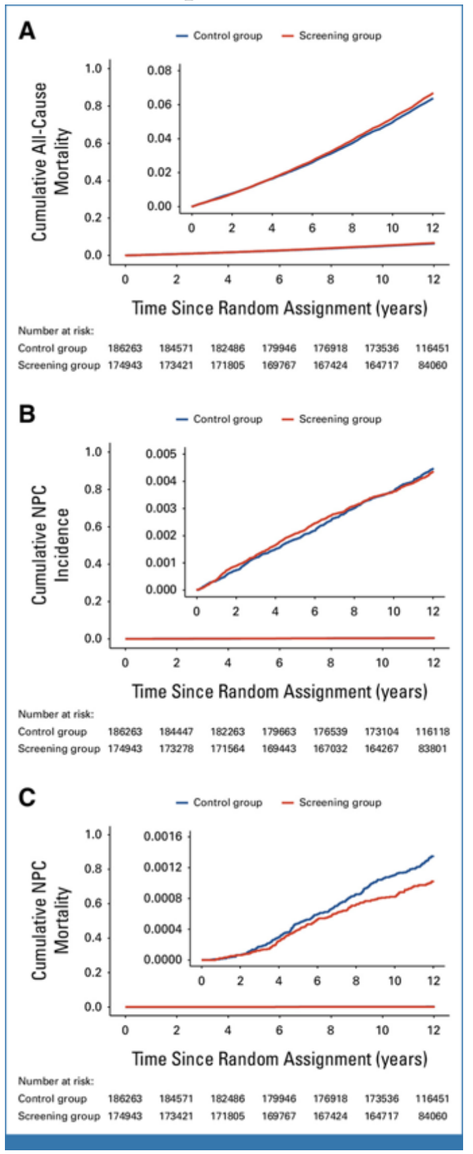
Impact of an Epstein-Barr Virus Serology-Based Screening Program on Nasopharyngeal Carcinoma Mortality: A Cluster-Randomized Controlled Trial

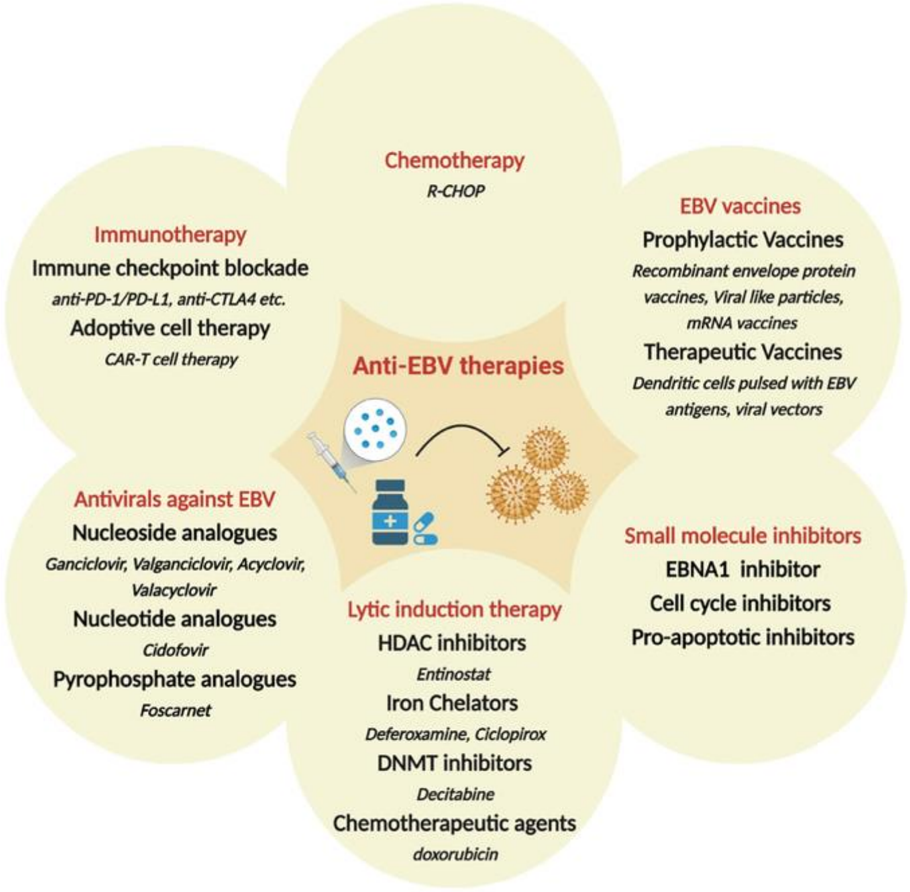
Authors: Wen-Jie Chen, MD, PhD, Xia Yu, MS, Yu-Qiang Lu, MD, Ruth M. Pfeiffer, PhD, Wei Ling, MD, Shang-Hang Xie, MS, Zhi-Cong Wu, PhD, ... SHOW ALL ..., and Su-Mei Cao, MD, PhD

AUTHORS INFO & AFFILIATIONS

J Clin Oncol 43, 22-31(2025) • Volume 43, Number 1 • DOI: 10.1200/JCO.23.01296



- **12 yıllık cluster-randomized çalışma**
- **VCA/EBNA1-IgA antikor taraması mortalitede %30'luk azalma**



doi: 10.3389/fimmu.2022.1059133

Science Bulletin 71 (2026) 838–849

Contents lists available at ScienceDirect

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Article

Global, regional, and national burden of hepatocellular carcinoma and contribution of nine modifiable risk factors across 185 countries/territories in 2022

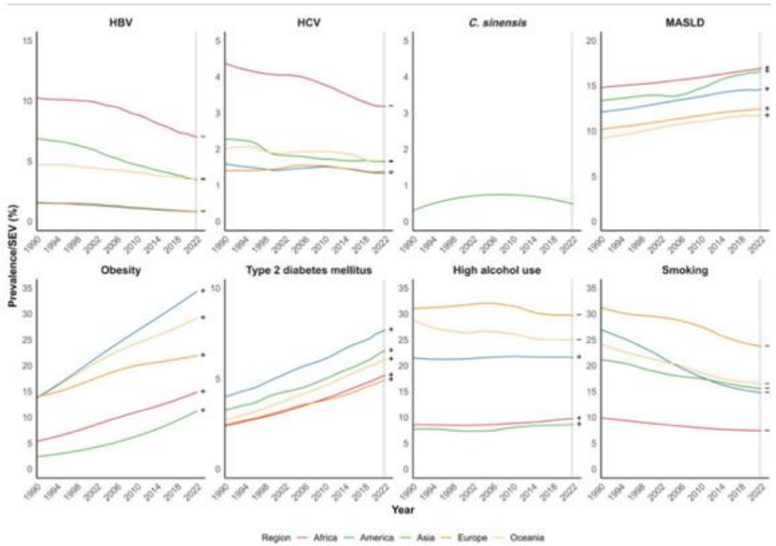


Table 2
Global population attributable fraction attributable to the nine risk factors for hepatocellular carcinoma in 2022, by 20-year latency period of exposure

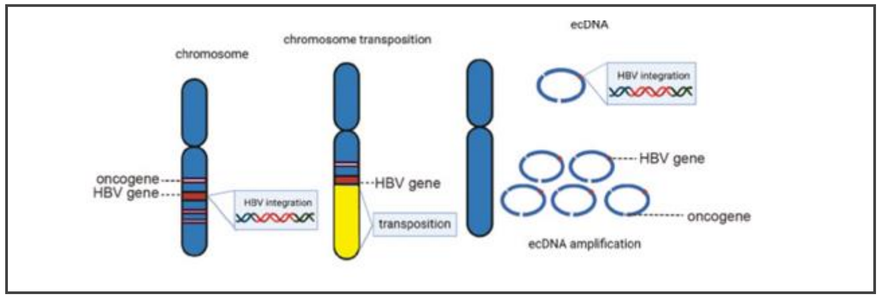
Risk factor	All		Male		Female	
	Attributable cases	PAF% (95% CI)	Attributable cases	PAF% (95% CI)	Attributable cases	PAF% (95% CI)
World						
All risk factors	536,571	78.4 (67.3–86.9)	402,066	80.1 (69.2–88.3)	134,505	73.7 (62.1–83.2)
Infection	450,952	65.9 (58.3–72.8)	339,673	67.6 (60.2–74.4)	111,279	61.0 (53.0–68.5)
HBV	390,750	57.1 (50.6–63.3)	298,048	59.4 (53.0–65.4)	92,702	50.8 (44.2–57.3)
HCV	128,902	18.8 (13.5–25.6)	93,005	18.5 (13.2–25.3)	35,897	19.7 (14.3–26.3)

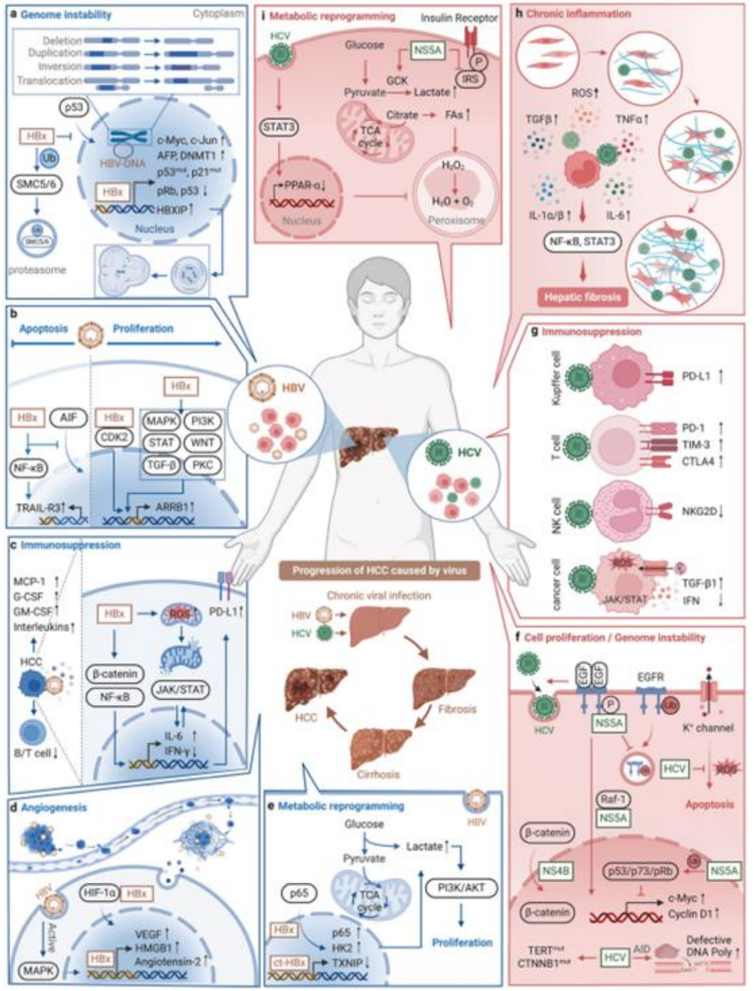
- **HBV ilişkili HCC en yüksek Moğolistan (%87,7) ve Vietnam'da (%87,4) gözlendi**
- **HBV enfeksiyonunun Asya'daki HCC vakalarının %69,7'sini ve Afrika'daki vakaların %44,9 unu oluşturmakta**
- **Tüm kıtalarda HBV ilişkili HCC azalma eğiliminde**

Table 2
Carcinogenic mechanisms of HBx.

Mechanisms	Function	Source of evidence
Activating a variety of transcription factors	Promoting NF-κB to inhibit TNF-α and Fas-mediated apoptosis	Cell experiments (hepatoma cell lines: HepG2 and Huh7)
	Stimulating the transcription factors CREB and ATF-2	Cell experiments (hepatoma cell line: HepG2)
	Disrupting the Smc5/6 complex and relieving transcriptional repression	Cell experiments (hepatoma cell lines: HepG2 and HepAD38) + animal experiments (human liver chimeric uPA-SCID mice)
Interacting with oncogenes and tumor suppressor genes	Inhibiting tumor suppressive genes (inhibiting *p53* and inactivating the *RB* gene)	Cell experiments (hepatocyte cell line: THLE-5b; hepatoma cell lines: Hep3B, SK-Hep-1, HepG2, Huh7, 97L, PLC/PRF/5, and SMMC7721) + animal experiments (HBx-transgenic mice and Balb/c nude mice) + clinical samples (paired HCC samples, $n = 51$)
	Activating oncogenes: the Ras-GTP complex	Cell experiments
Stimulating cytoplasmic signaling pathways	Activating the mTOR signaling pathway	Cell experiments (hepatoma cell lines: HepG2, Hep3B, Huh7, and Hep2.2.15) + animal experiments (DEN-induced HCC mouse model and spontaneous HCC mouse model) + clinical samples (paired HCC samples, $n = 80$)
	Affecting the Fas/FasL signaling pathway	Cell experiments (hepatoma cell lines: HepG2, SNU-354, SNU-368, SNU-387, SNU-398, and SNU-423) + animal experiments (HBx homozygote transgenic mice) + clinical samples (paired HCC samples, $n = 15$)
Interacting with noncoding RNAs	Affecting miRNAs, such as miR-122 and miR-152	Cell experiments (hepatocyte cell line: LO2; hepatoma cell lines: Hepa1-6, HepG2, HepG2.2.15, and Huh7) + animal experiments (HBx-transgenic mice) + clinical samples (chronic hepatitis B samples, $n = 22$; severe chronic hepatitis B samples, $n = 19$; normal liver tissues, $n = 10$; paired HCC samples, $n = 20$)
	Affecting lncRNA, such as lncRNA DLEU2 and LINC01431X	Cell experiments (hepatoma cell lines: HepG2, HLCZ01, HepAD38, Huh7, and HepG2.2.15) + animal experiments (hydrodynamic injection mouse model) + clinical samples (para-tumor tissues, $n = 41$)

Abbreviations: ATF-2, activating transcription factor-2; CREB, cAMP response element binding protein; DEN, diethylnitrosamine; HBx, hepatitis B virus X; H cellular carcinoma; lncRNA, long-stranded noncoding RNA; miRNA, microRNA; mTOR, mammalian target of rapamycin; NF-κB, nuclear factor-kappa B; RB, reti Smc5/6, structural maintenance of chromosomes 5/6; TNF-α, tumor necrosis factor-alpha.





doi:10.1038/s41392-025-02197-9
doi:10.1016/j.livres.2025.09.002

RESEARCH ARTICLE · Volume 78, Issue 3, P534-542, March 2023 · *Open Access* Download Full Issue

Hepatocellular carcinoma risk in patients with chronic hepatitis B receiving tenofovir- *vs.* entecavir-based regimens: Individual patient data meta-analysis

Won-Mook Choi [1,†] · Terry Cheuk-Fung Yip [2,†] · Grace Lai-Hung Wong [2] · ... · Jung Woo Shin [15] · Yao-Hsu Yang [20,21] · Young-Suk Lim [1] ... Show more

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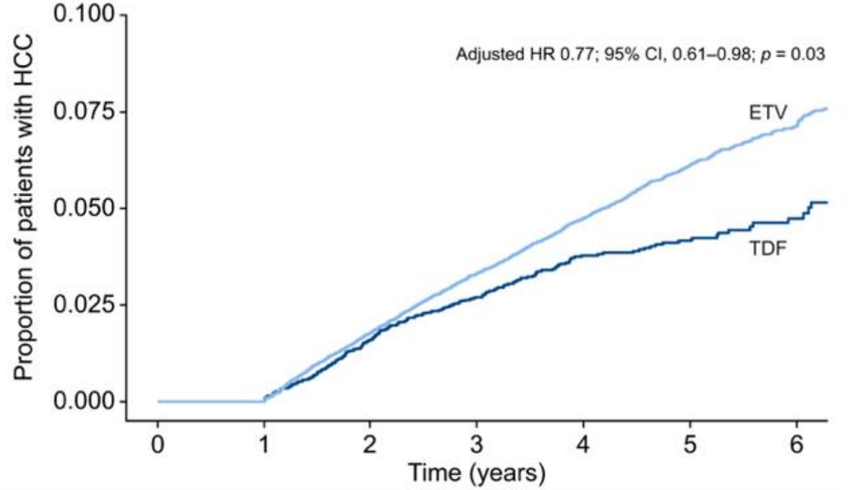
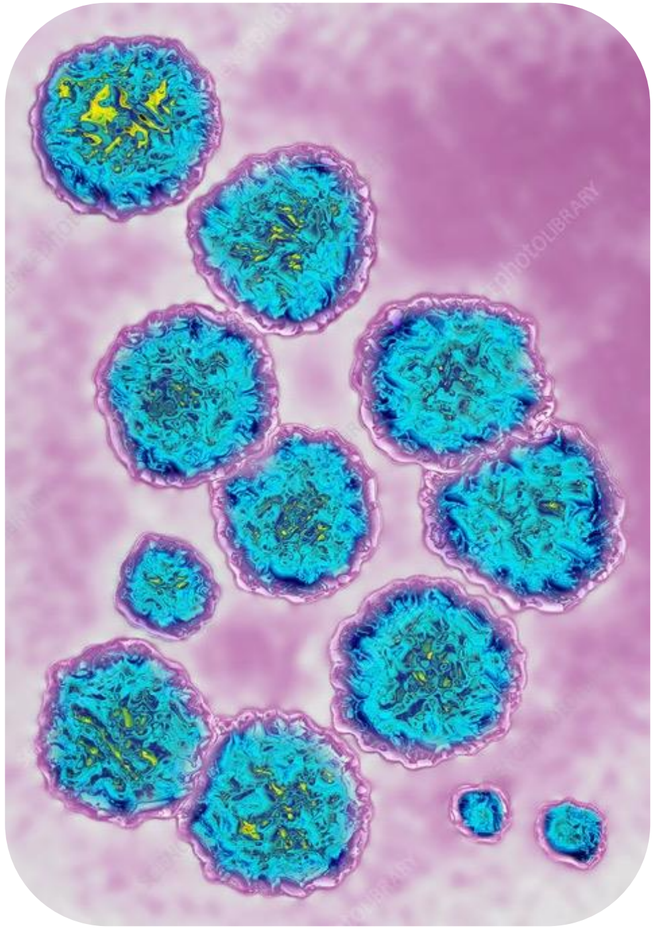
- **TDF kullanan hastalarda HCCriskidüşüktü (HR 0,77; %95 CI 0,61–0,98)**
- **Özellikle ≥50 yaş, erkek, HBeAg pozitif ve diyabeti olmayan hastalarda daha yüksek**



N° at risk (%)							
ETV	35,960 (100)	35,960 (100)	29,679 (83)	24,642 (69)	19,749 (55)	13,046 (36)	4,383 (12)
TDF	6,979 (100)	6,979 (100)	5,792 (83)	4,387 (63)	2,927 (42)	1,730 (25)	751 (11)

Hepatit C Virus

HCV



Global burden of cancer attributable to infections in 2018: a worldwide incidence analysis

Catherine de Martel, Damien Georges, Freddie Bray, Jacques Ferlay, Gary M Clifford

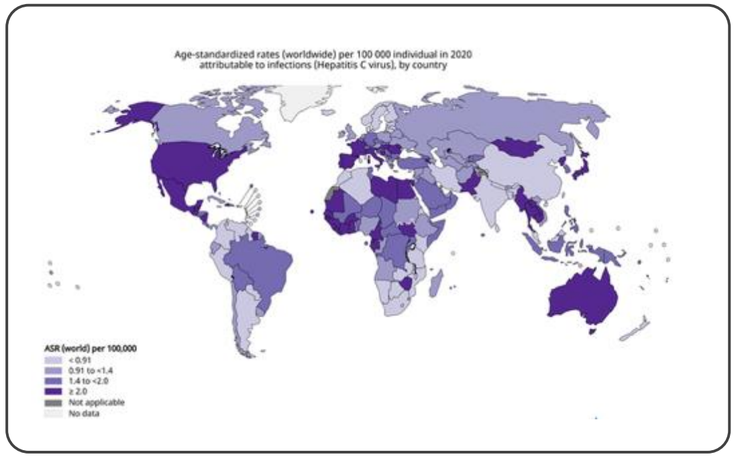
Summary

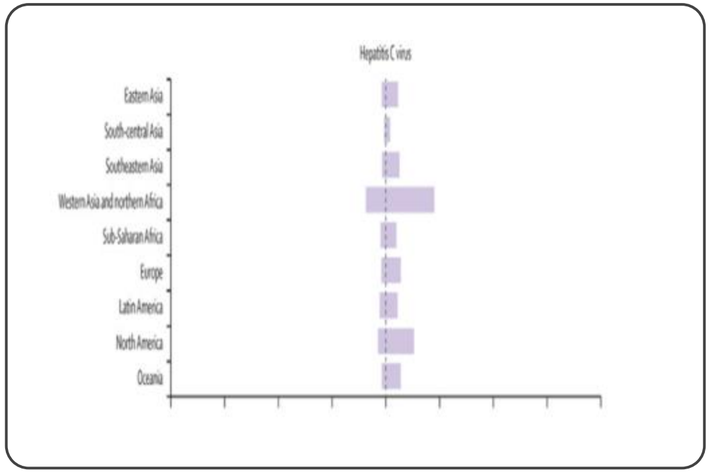
Background Infectious pathogens are strong and modifiable causes of cancer. The aim of this study was to improve estimates of the global and regional burden of infection-attributable cancers to inform research priorities and facilitate prevention efforts

Lancet Glob Health 2020; 8: e180–90

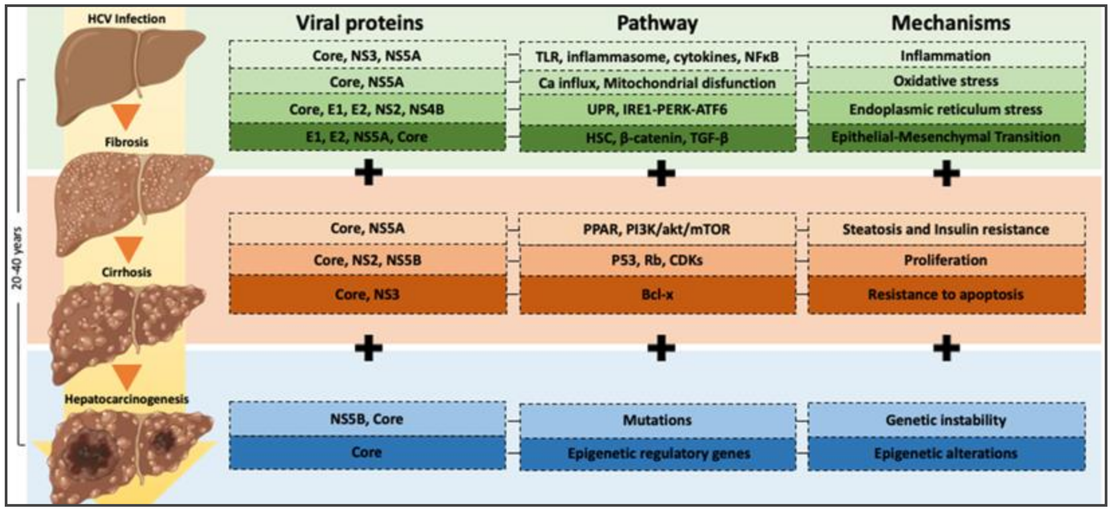
Published Online

Hepatitis C virus						
Hepatocellular carcinoma	490 000	100 000	170 000	40 000	660 000	140 000
Other non-Hodgkin lymphoma	260 000	8700	210 000	7200	480 000	16 000

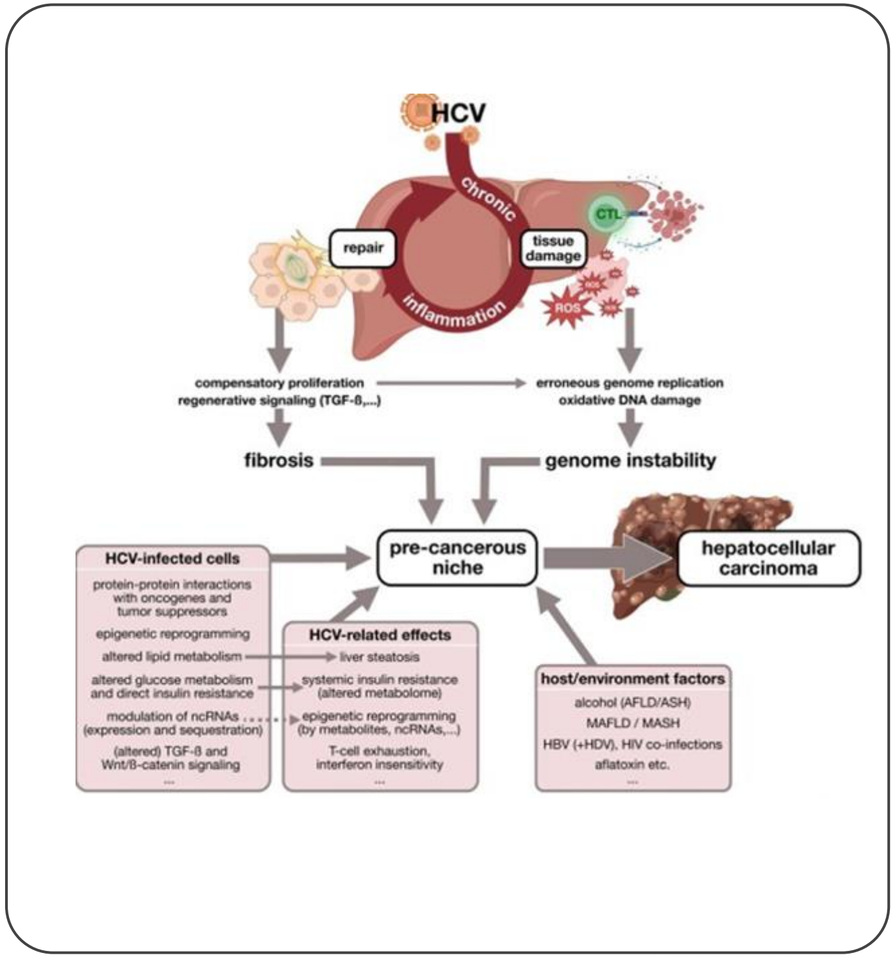




- **HCV ilişkili kanser insidansı: 1,7/100.000 (büyük bölümü HCC)**
- **Karaciğer kanserlerinin %29,1'i HCV kaynaklı**
- **Yükün %80'i: Çin, Hindistan, Pakistan, Rusya, ABD**
- **En yüksek ASIR: Kuzey Afrika (12,0); Japonya (4,1) ve ABD (3,6) ortalamanın üzerinde**
- **~16.000 NHL vakası HCV'ye atfedilebilir**

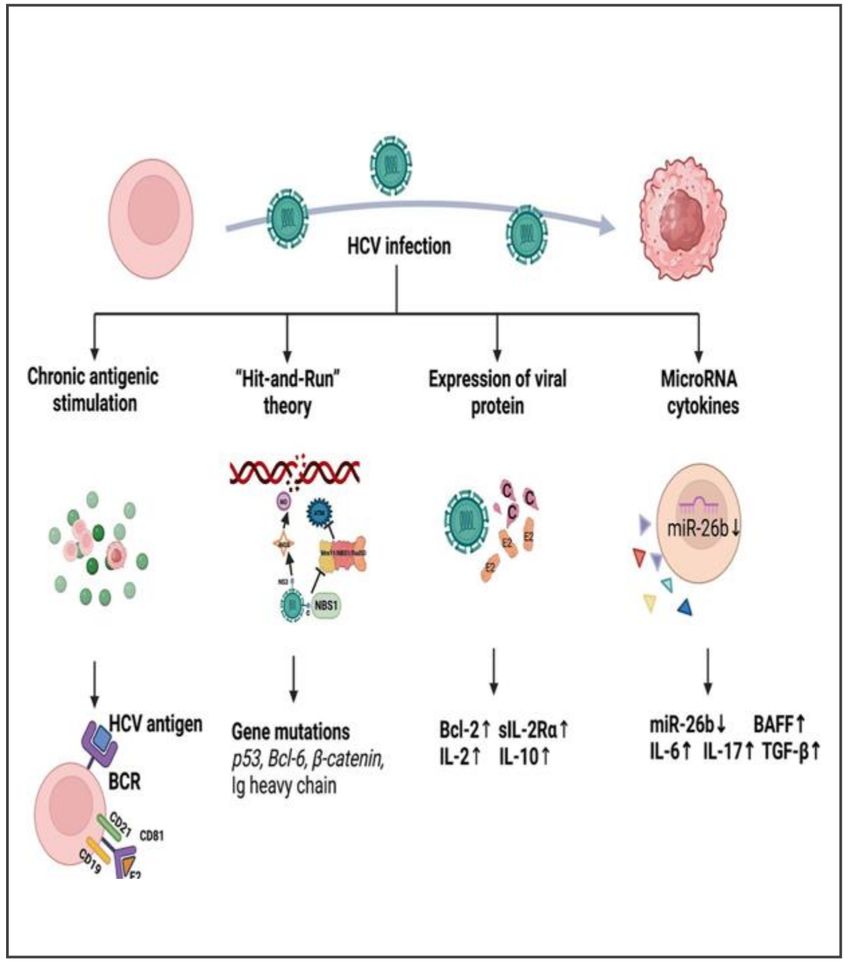


- HCV: core, NS3, NS4B, NS5A, NS5B → onkojenik etkiler
- Core protein: NF-κB, IL-6, STAT3 aktivasyonu
- NS proteinler: apoptoz ↓, proliferasyon ↑
- Tümör baskılayıcı proteinlerle etkileşim → genomik instabilite
- Hücre döngüsü bozulur, onkogen ekspresyonu ↑
- Kronik inflamasyon → mutasyon ve dönüşüm



doi:10.3390/v14122762

- HCV ile lenfoma gelişiminde temel mekanizma kronik antijenik uyarımı, B hücre aktivasyonu ve proliferasyon
- HCV, proto-onkogen ve onkogenlerde değişiklikler oluşturarak mutasyon fenotipi indükler
- Bu süreç B hücrelerinde kademeli onkojenik dönüşüm
- Etki, virüs temizlendikten sonra da devam edebilir



doi.org/10.3389/fimmu.2024.1361009

Kalıcı virolojik yanıt ve HCC

- Kalıcı virolojik yanıt sonrası HCC riski azalır ancak tamamen ortadan kalkmaz
- Sirozlu hastalarda en yüksek risk ilk 1–2 yılda, 2–6 yıl arasında belirgin azalma
- 6 yıl sonrasında riskte ek azalma olmaz, plato eğilimi izlenir
- Sirozu olmayanlarda HCC riski düşüktür
- Bu grupta SVR süresi ile HCC riski arasında anlamlı ilişki yoktur
- Yüksek FIB-4, yüksek komorbidite ve genotip 3 → artmış HCC riski

▸ Aliment Pharmacol Ther. Author manuscript; available in PMC: 2025 Feb 1.

Published in final edited form as: Aliment Pharmacol Ther. 2023 Nov 13;59(3):361–371. doi: 10.1111/apt.17802

Hepatocellular carcinoma risk decreases as time accrues following hepatitis C virus eradication

Philip Vutien [1,2], Nicole J Kim [1,2], Andrew M Moon [3,4], Kay M Johnson [5,6], Kristin Berry [7], Pamela K Green [7], George N Ioannou [1,2,7]

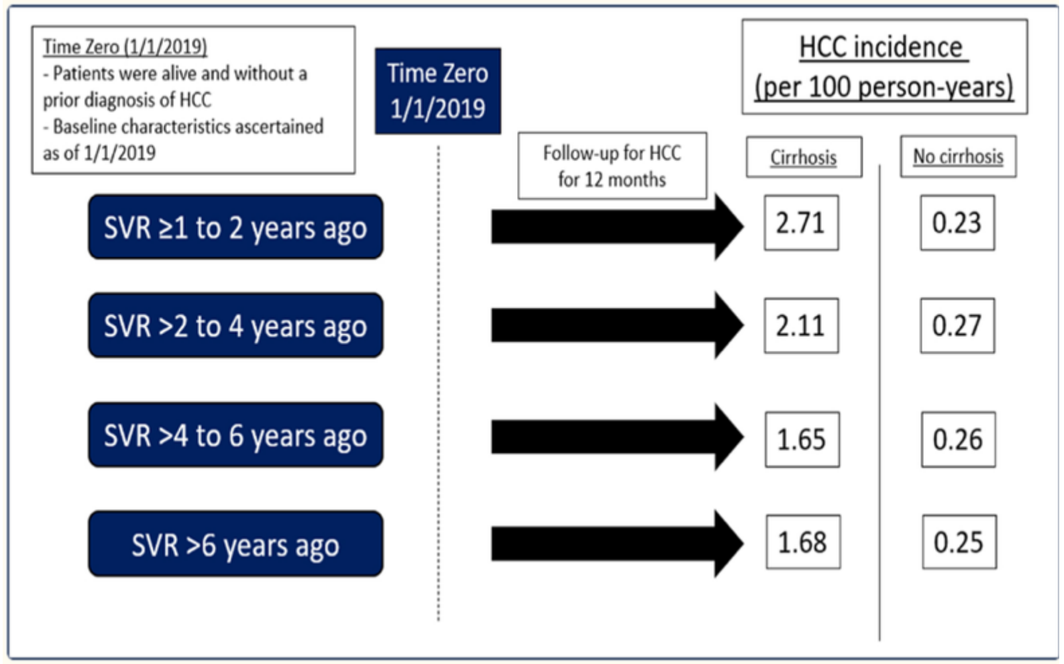
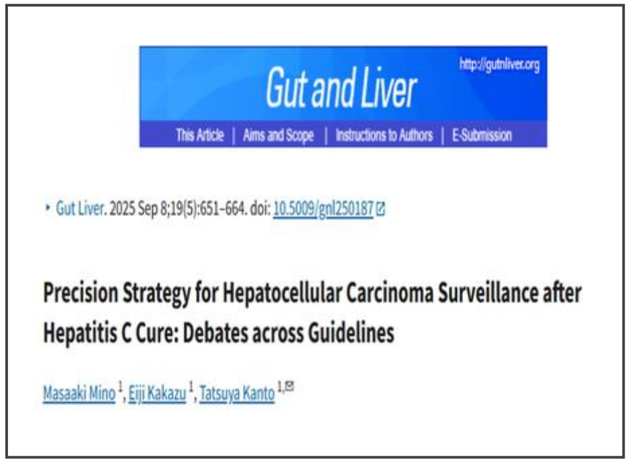


Table 3. Comparative Summary of Post-SVR HCC Surveillance Guidelines from Major Hepatology Societies

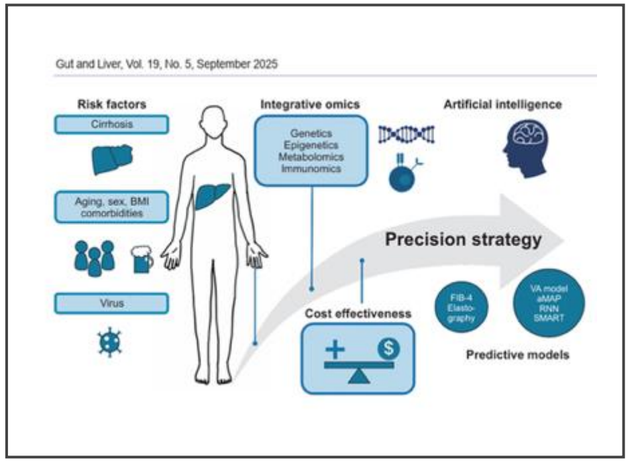
	EASL	AASLD	APASL	KASL	TASL	JSH*
Target group	F3-F4	F4	F0-F4	F3-F4	F0-F4	F0-F4
Screening interval	Every 6 mo indefinitely	Every 6 mo indefinitely	F0-F2: every 6 mo for 2 yr, then annually F3-F4: every 6 mo	Every 6 mo	F0-1 with HCC risk factors and F2: every 6–12 mo F3-4: every 3–6 mo	Every 6 mo for chronic hepatitis, every 3–4 mo for cirrhosis, indefinitely
Modality	US with AFP	US with AFP	US with AFP, PIVKA-II, AFP-L3	US with AFP	NA	US with AFP, PIVKA-II, AFP-L3
Advantages	Clear recommendations for advanced fibrosis stages	Clear recommendations for cirrhosis stages	Comprehensive surveillance, tailored to fibrosis stage	Clear recommendations for advanced fibrosis stages	Continuous focus on early detection depending on the HCC risks	Continuous focus on early detection
Disadvantages	Limited guidance for early-stage fibrosis	Limited guidance for early-moderate stage fibrosis	Repeated high costs	Limited guidance for early-stage fibrosis	Unclear cost-effectiveness	Unclear cost-effectiveness
Publication year	2025[127], 2024[133]	2023[128]	2019[125,126]	2016[130], 2022[131]	2020[132]	2021[129]



- Mevcut tarama önerileri heterojendir ve çoğunlukla fibroz derecesine dayanır
- Risk prediksiyon modelleri, bireyselleştirilmiş izlem için umut vadeder
- Gelecek yaklaşım: klinik, genomik, metabolik ve immün verilerin entegrasyonu

Kılavuz farklılıkları

- AASLD: sadece siroz hastalarında tarama
- EASL: F3 fibroz + sirozda tarama
- APASL: tüm hastalarında tarama



microorganisms MDPI

Review

Extrahepatic Cancer Risk in Patients with Hepatitis C Virus Infection Treated with Direct-Acting Antivirals

Joji Tani [1,*], Tsutomu Masaki [2], Kyoko Oura [1], Tomoko Tadokoro [1], Asahiro Morishita [1] and Hideki Kobara [1]

Table 2. Comparison of extrahepatic cancer risk.

Treatment Strategy	Overall Extrahepatic Cancer Risk	Specific Cancer Types	Relative Risk (95% CI)	Follow-Up Period	Key References
HCV—No Treatment	Increased	Non-Hodgkin Lymphoma Multiple Myeloma Head and Neck	2.24 (1.80–2.78) 1.97 (1.31–2.96) 1.56 (1.32–1.84)	Varied	Mahale et al. (2017)
HCV—Traditional (IFN/Ribavirin)	Moderately Decreased	Non-Hodgkin Lymphoma	0.73 (0.55–0.96)	Median 5.6 years	Kawamura et al. (2017)
		All Extrahepatic	0.81 (0.66–0.99)		
HCV—DAA	Potentially Decreased	All Extrahepatic	0.84 (0.73–0.96)	Median 33.4 months	Carrat et al. (2019)
		Hematological	0.64 (0.38–1.07)		
		Non-Hematological	0.86 (0.74–0.99)		

CI: confidence interval. HCV: hepatitis C virus. DAA: direct-acting antivirals.

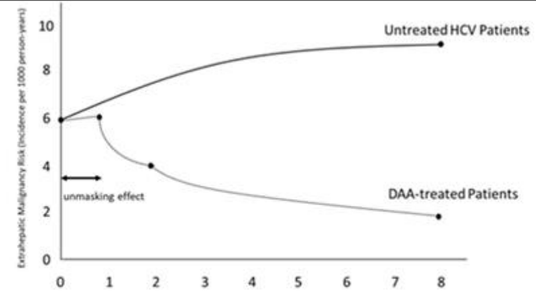
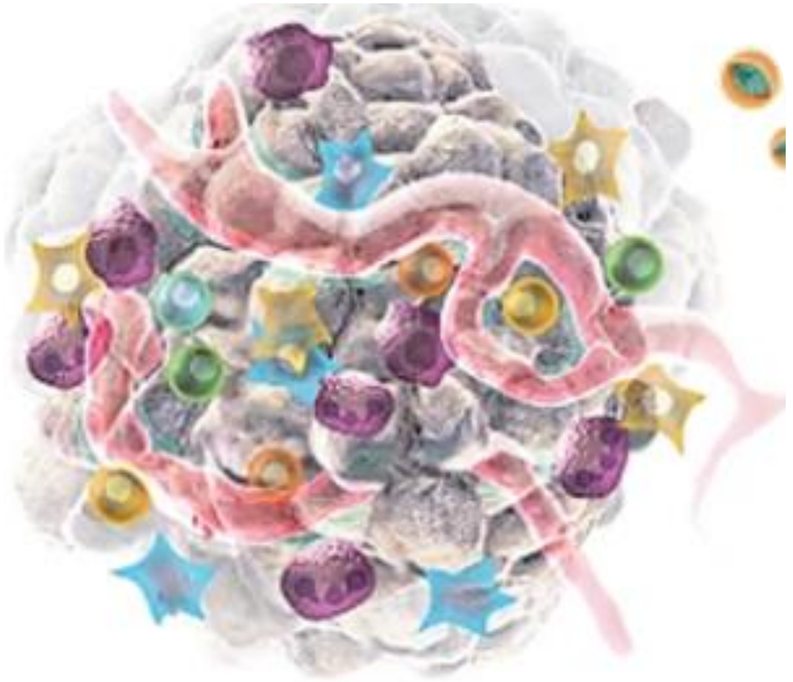


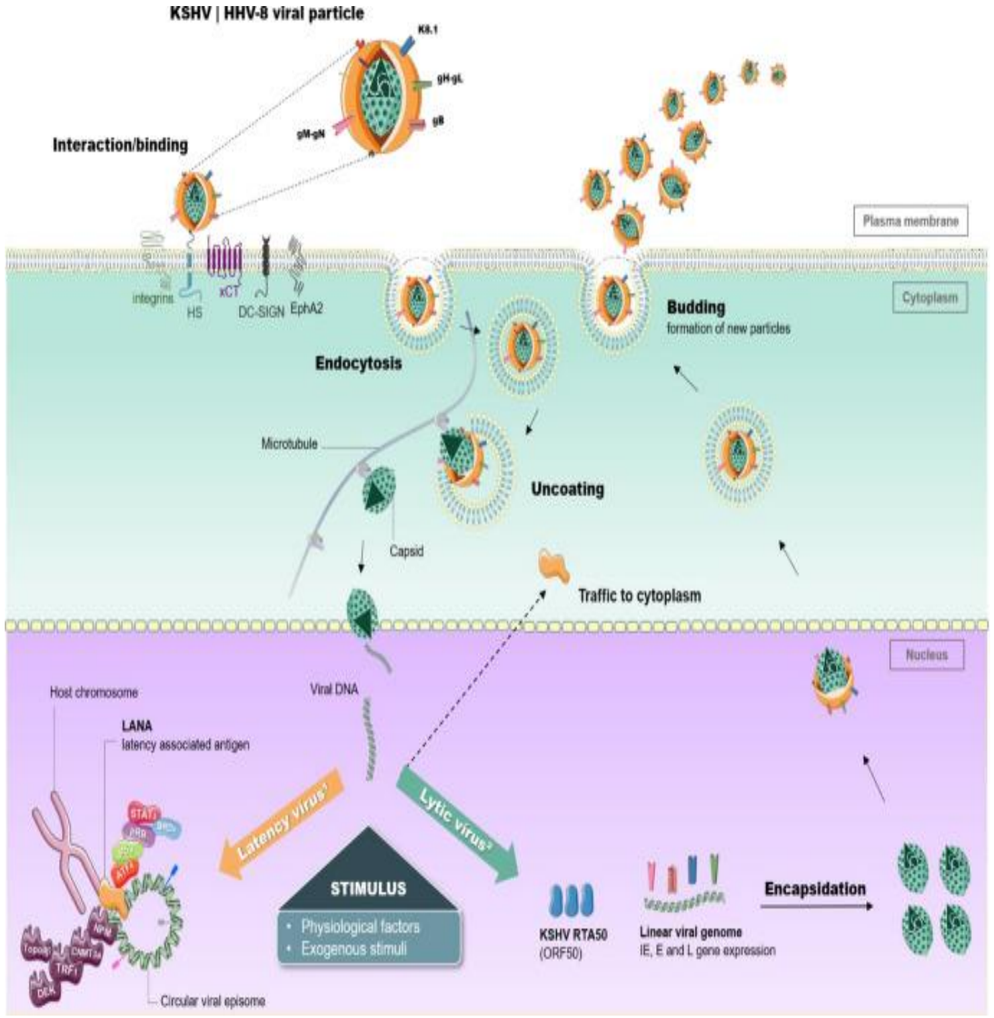
Figure 2. Impact of DAA treatment on extrahepatic malignancy risk. While short-term data may have shown an apparent increase in cancer diagnoses due to enhanced detection, long-term evidence supports a reduced risk of extrahepatic malignancies in successfully DAA-treated patients. HCV: hepatitis C virus. DAA: direct-acting antivirals.

- **DEA tedavisinin karaciğer dışı malignite riski üzerindeki etkisi tartışmalı**
- **Kısa vadede, artan tanı nedeniyle kanser insidansı yüksek görünür**
- **Uzun vadede, DEA ile tedavi edilenlerde karaciğer dışı malignite riski azalır**

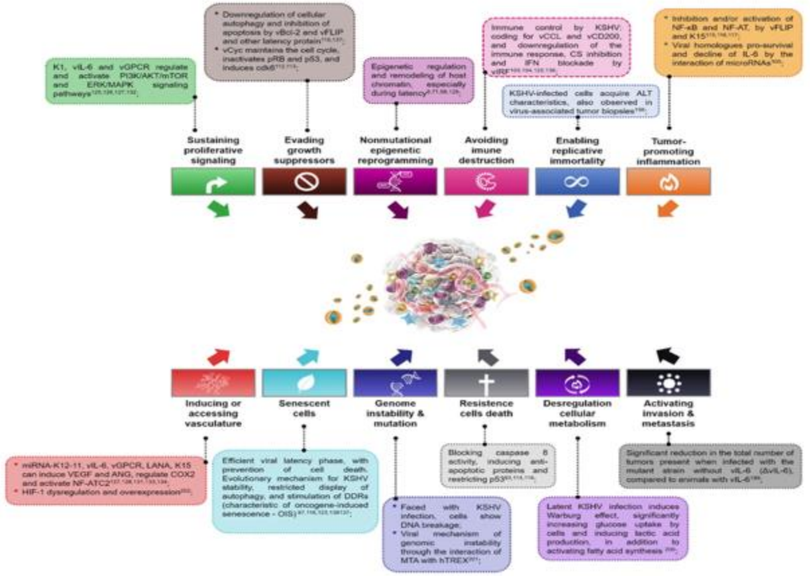
Kaposi's Sarcoma-associated Herpesvirus

KSHV

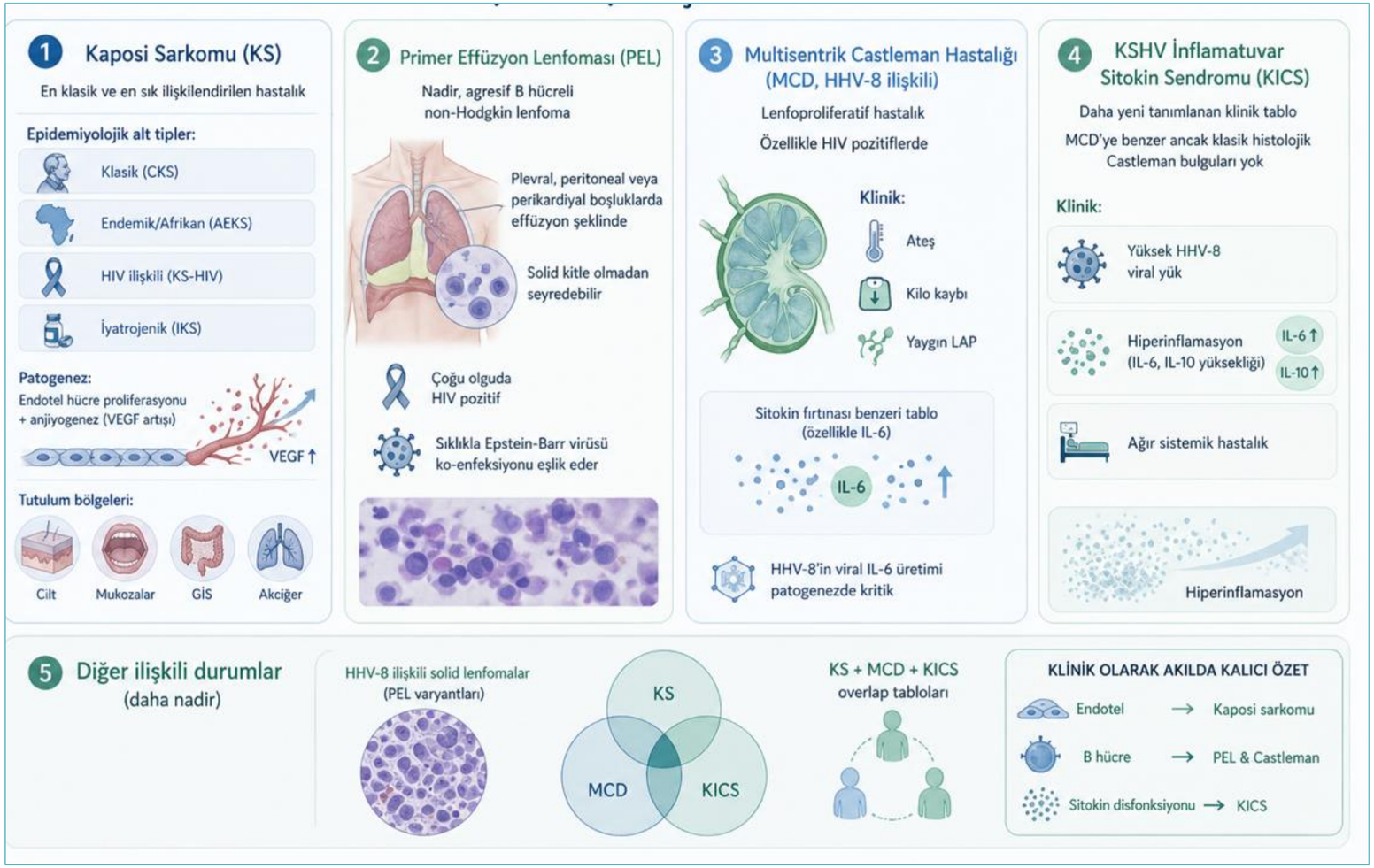


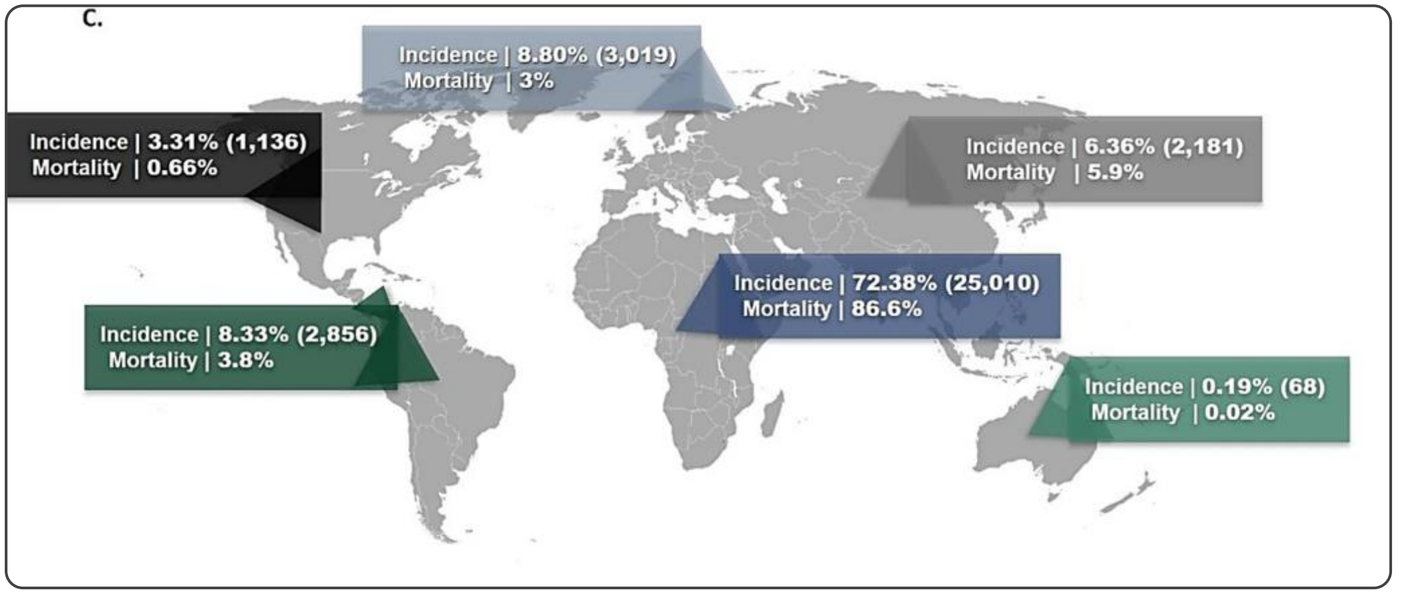


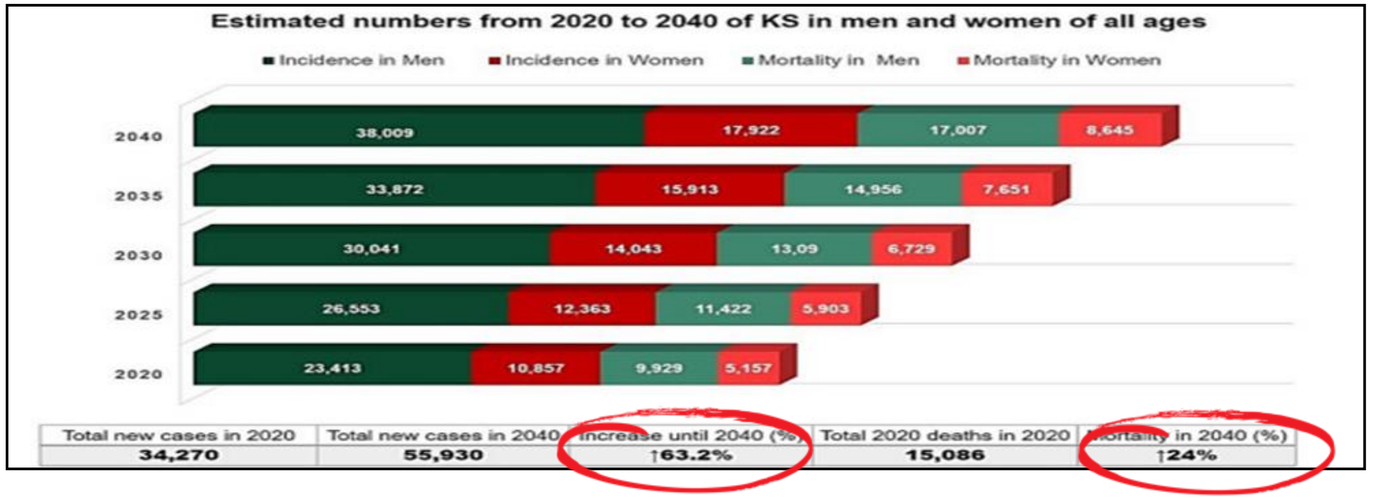
- **LANA, KSHV enfeksiyonunun modülasyonunda önemli bir onkoprotein**
- **HIV varlığında TAT**

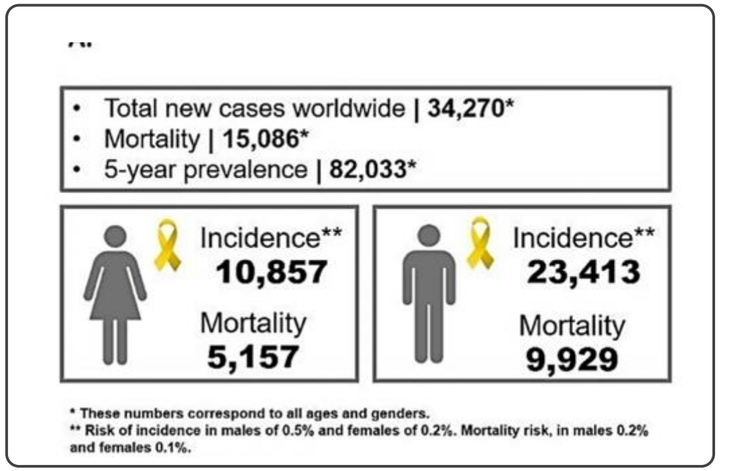


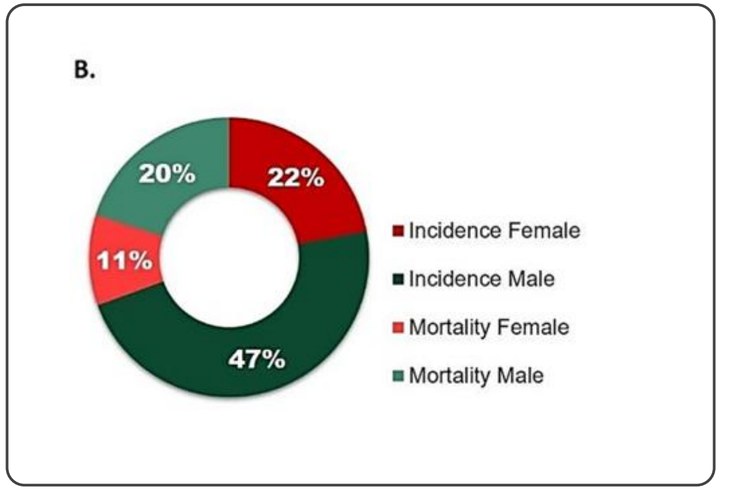
doi: 10.1007/s00203-025-04706-4


1 Kaposi Sarkomu (KS)
En klasik ve en sık ilişkilendirilen hastalık
Epidemiyolojik alt tipler:
Klasik (CKS)
Endemik/Afrikan (AEKS)
HIV ilişkili (KS-HIV)
İyatrojenik (IKS)
Patogenez:
Endotel hücre proliferasyonu
+ anjiyogenez (VEGF artışı)
VEGF ↑
Tutulum bölgeleri:
Cilt
Mukozalar
GİS
Akciğer
2 Primer Effüzyon Lenfoması (PEL)
Nadir, agresif B hücreli
non-Hodgkin lenfoma
Plevral, peritoneal veya
perikardiyal boşluklarda
effüzyon şeklinde
Solid kitle olmadan
seyredebilir
Çoğu olguda
HIV pozitif
Sıklıkla Epstein-Barr virüsü
ko-enfeksiyonu eşlik eder
3 Multisentrik Castleman Hastalığı
(MCD, HHV-8 ilişkili)
Lenfoproliferatif hastalık
Özellikle HIV pozitiflerde
Klinik:
Ateş
Kilo kaybı
Yaygın LAP
Sitokin fırtınası benzeri tablo
(özellikle IL-6)
IL-6
HHV-8'in viral IL-6 üretimi
patogenezde kritik
4 KSHV İnflamatuvar
Sitokin Sendromu (KICS)
Daha yeni tanımlanan klinik tablo
MCD'ye benzer ancak klasik histolojik
Castleman bulguları yok
Klinik:
Yüksek HHV-8
viral yük
Hiperinflamasyon
(IL-6, IL-10 yüksekliği)
IL-6 ↑
IL-10 ↑
Ağır sistemik hastalık
Hiperinflamasyon
5 Diğer ilişkili durumlar
(daha nadir)
HHV-8 ilişkili solid lenfomalar
(PEL varyantları)
KS
MCD
KICS
KS + MCD + KICS
overlap tabloları
KLİNİK OLARAK AKILDA KALICI ÖZET
Endotel → Kaposi sarkomu
B hücre → PEL & Castleman
Sitokin disfonksiyonu → KICS









doi: 10.1007/s00203-025-04706-4

Global patterns and trends in Kaposi sarcoma incidence: a population-based study

Leiwen Fu, Tian Tian*, Bingyi Wang*, Zhen Lu*, Yanxiao Gao*, Yinghui Sun, Yi-Fan Lin, Weijie Zhang, Yuwei Li, Huachun Zou*

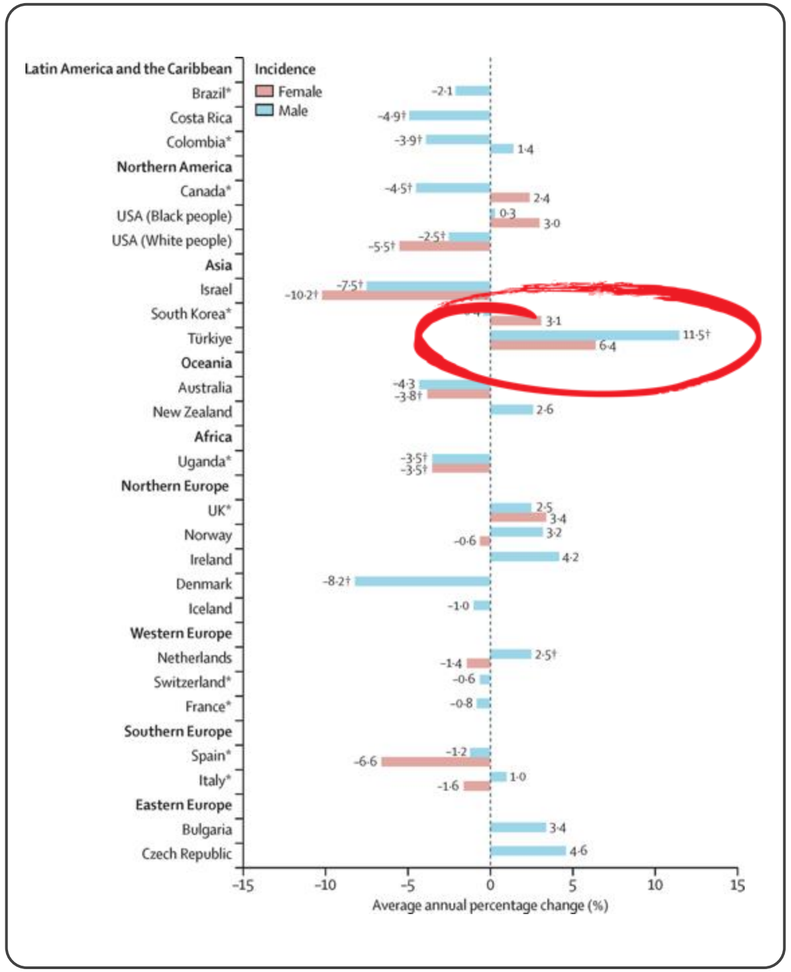
Summary

Background Kaposi sarcoma is a rare, possibly angioproliferative, tumour. Kaposi sarcoma is one of the most common cancers in people living with HIV and poses a serious public health challenge in regions with high HIV burden. We aim to describe global patterns and population-wide trends in the burden of Kaposi sarcoma.

Lancet Glob Health 2023; 11: e1566–75

See Comment page e1479

- **Birçok popülasyonda erkeklerde ve kadınlarda Kaposi sarkoması insidansının azalmakta**
- **Türkiye'de hem erkek hem kadın cinsiyette artış eğilimi (%11/%6,4)**



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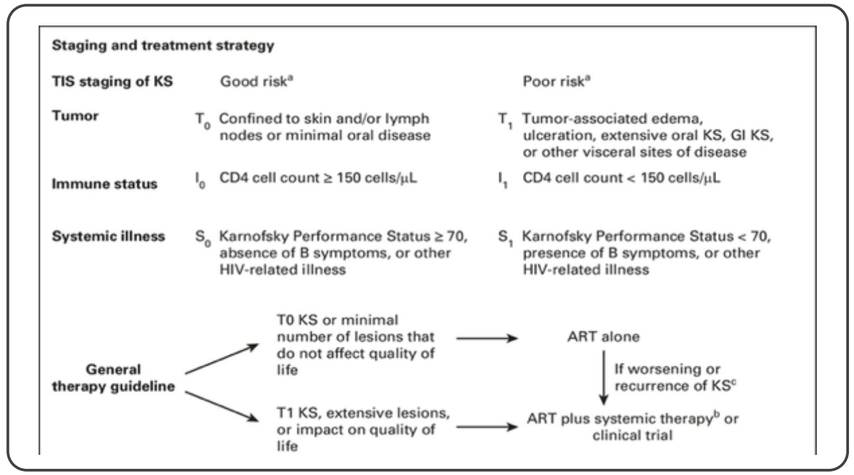
Oncologic Treatment of HIV-Associated Kaposi Sarcoma 40 Years on

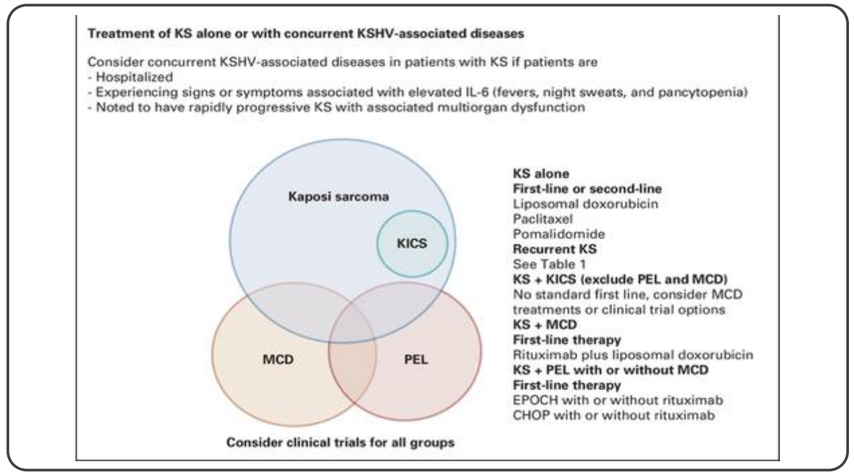
Authors: Ramya Ramaswami, MBBS, MPH , Kathryn Lurain, MD, MPH , and Robert Yarchoan, MD AUTHORS INFO & AFFILIATIONS

J Clin Oncol **40**, 294-306(2022) • Volume 40, Number 3 • DOI: 10.1200/JCO.21.02040



Staging and treatment strategy

TIS staging of KS	Good risk[a]	Poor risk[a]
Tumor	T_0 Confined to skin and/or lymph nodes or minimal oral disease	T_1 Tumor-associated edema, ulceration, extensive oral KS, GI KS, or other visceral sites of disease
Immune status	I_0 CD4 cell count ≥ 150 cells/μL	I_1 CD4 cell count < 150 cells/μL
Systemic illness	S_0 Karnofsky Performance Status ≥ 70, absence of B symptoms, or other HIV-related illness	S_1 Karnofsky Performance Status < 70, presence of B symptoms, or other HIV-related illness



Treatment of KS alone or with concurrent KSHV-associated diseases

Consider concurrent KSHV-associated diseases in patients with KS if patients are
- Hospitalized
- Experiencing signs or symptoms associated with elevated IL-6 (fevers, night sweats, and pancytopenia)
- Noted to have rapidly progressive KS with associated multiorgan dysfunction

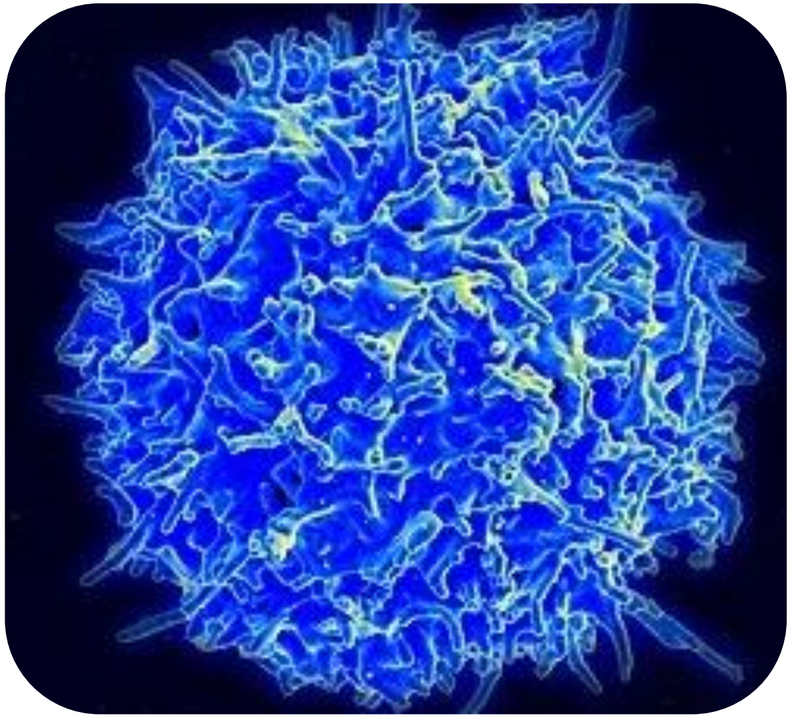
KS alone
First-line or second-line
Liposomal doxorubicin
Paclitaxel
Pomalidomide
Recurrent KS
See Table 1
KS + KICS (exclude PEL and MCD)
No standard first line, consider MCD treatments or clinical trial options
KS + MCD
First-line therapy
Rituximab plus liposomal doxorubicin
KS + PEL with or without MCD
First-line therapy
EPOCH with or without rituximab
CHOP with or without rituximab

Consider clinical trials for all groups

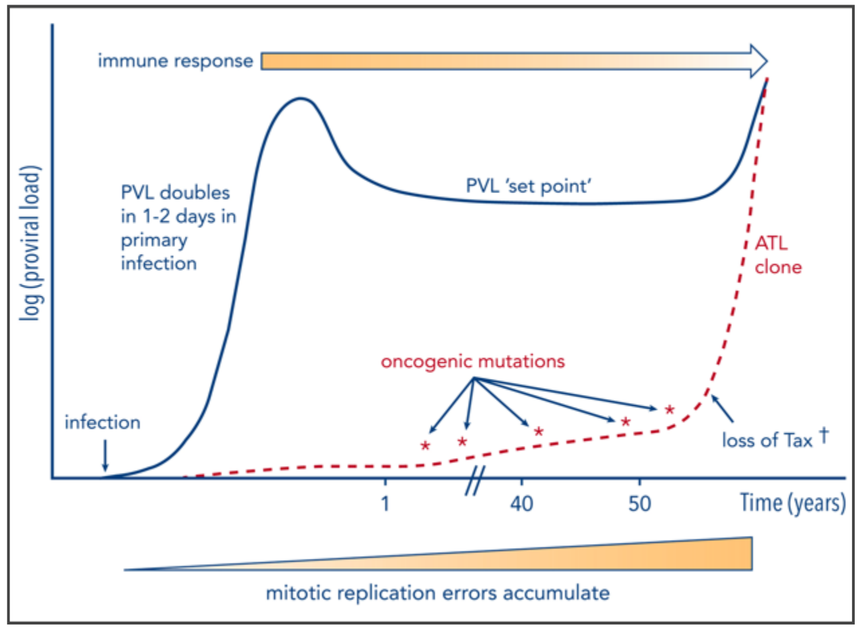
- **ART şart: Erken evrede tek başına yeterli olabilir**
- **İleri hastalık: Kemoterapi (liposomal doksorubisin, paklitaksel)**
- **Yeni seçenekler: İmmünoterapi ve immünomodülatörler**

Human T-cell Lymphotropic Virus Type 1

HTLV1

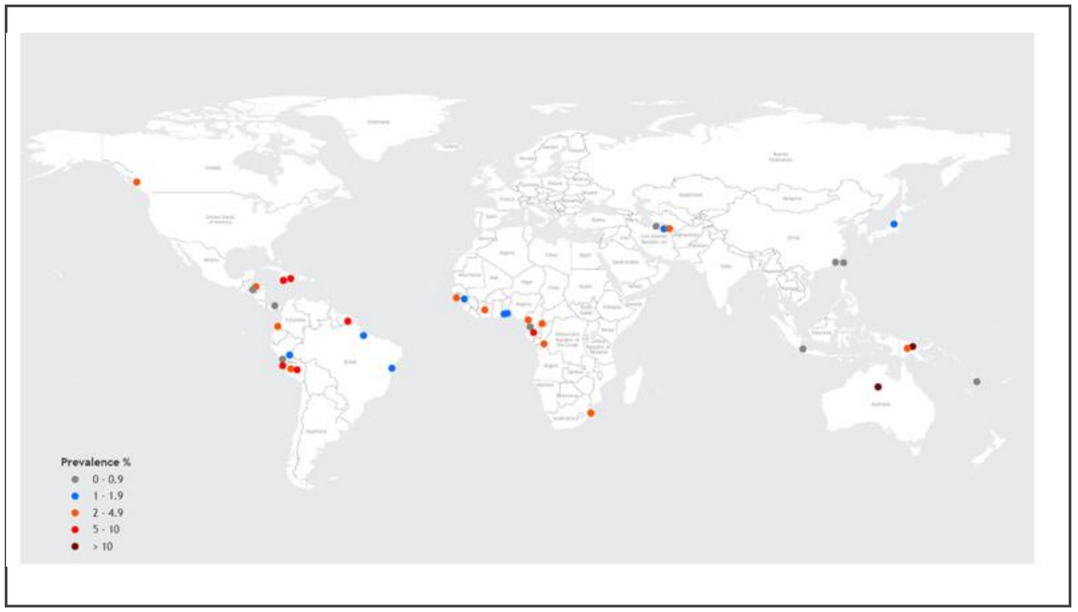


- ATL, kademeli somatik mutasyon birikimiyle gelişir
- Tax ve HBZ, onkogenezde temel rol oynar
- Ana mekanizma: uzun ömürlü T hücrelerinde mitotik replikasyon hataları

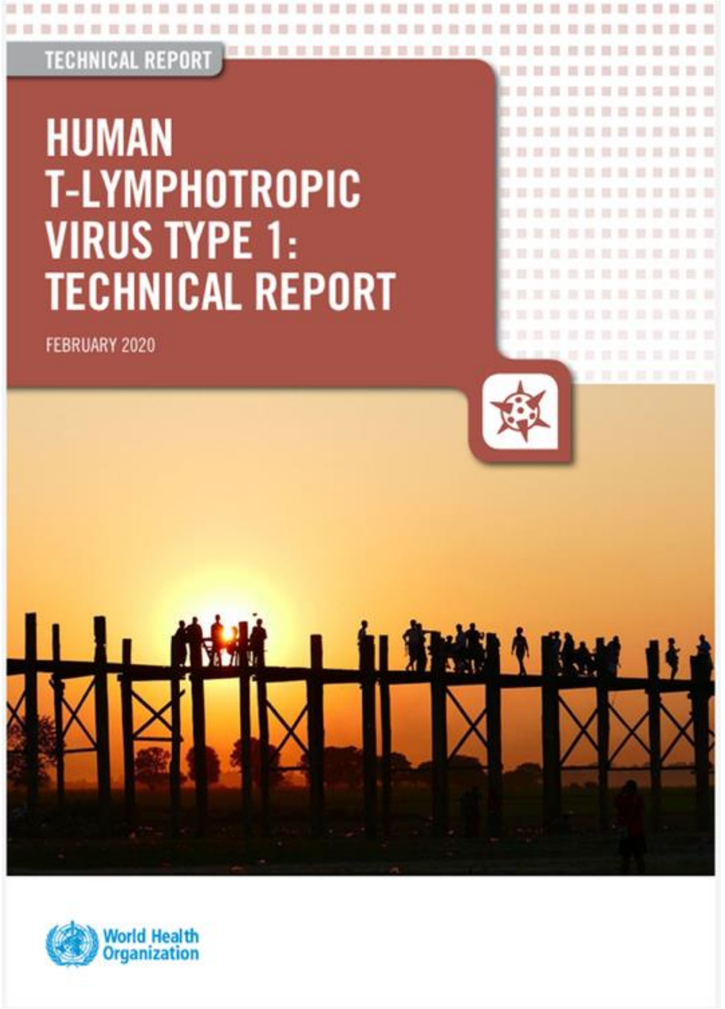


HTLV Type	Region	Key Countries/Areas	Prevalence Range (General Population/Specific Groups)	Data Collection Period
HTLV-1	Southwestern Japan	Kyushu, Okinawa	Blood donors: 1% (Hokkaido) to >6% (Kyushu, Okinawa)	2006–2016
	Sub-Saharan Africa	Gabon, DRC, Nigeria, Ghana, Guinea-Bissau	Adults: 0.3–3%; Older women (Gabon/DRC): 10–25%; Pregnant women (West Africa): 0.2–7.7%	early 2000s–2010s
	South America	Peru, Colombia, French Guiana, Brazil	Blood donors (Brazil): 0.04–1%	2000s–2010s
	Caribbean Area	Jamaica, Haiti	Jamaica (mean): 6.1%; Pregnant women (Haiti): 2.2–4.2%	1990s–2000s
	Middle East	Iran (Mashad region)	Adults: 0.77–3%	2003–2011
	Australo-Melanesia	Central Australia, PNG, Solomon Islands	Aboriginal Australians: up to 44%; Tribes: 1.2–3%	1990s–2000s
	Southeastern USA		Prevalence in blood donors, regional variations	2007–2015
HTLV-2	Indigenous populations of the Americas	Brazil (Amazon), Panama, USA	Kayapó: up to 41.2%; Native American tribes: up to 13%; Mexico: 0.23%	2000s–2010s
	People who inject drugs (PWID)	North America, Europe	Estimated prevalence: 20% (USA)	1990s–2010s
	Some Indigenous people in Africa	Cameroon, DRC (Pygmy populations)	Detected in Pygmy populations	2000s
	USA		Blood donors: HTLV-2 more common than HTLV-1; overall prevalence: 0.016%	2007–2015

doi:10.3390/v17050664



- Güneybatı Japonya
- Karayip havzası
- Güney Amerika
- Batı Afrika



North Clin Istanb. 2025 Nov 5;12(5):653–660. doi: 10.14744/nci.2025.23571

Global epidemiology of HTLV: Under-reported and under-studied regions

Hossein Mardnaybin [1], Mehmet Demirci [2], Hayriye Kirkoyun Uysal [3,✉]

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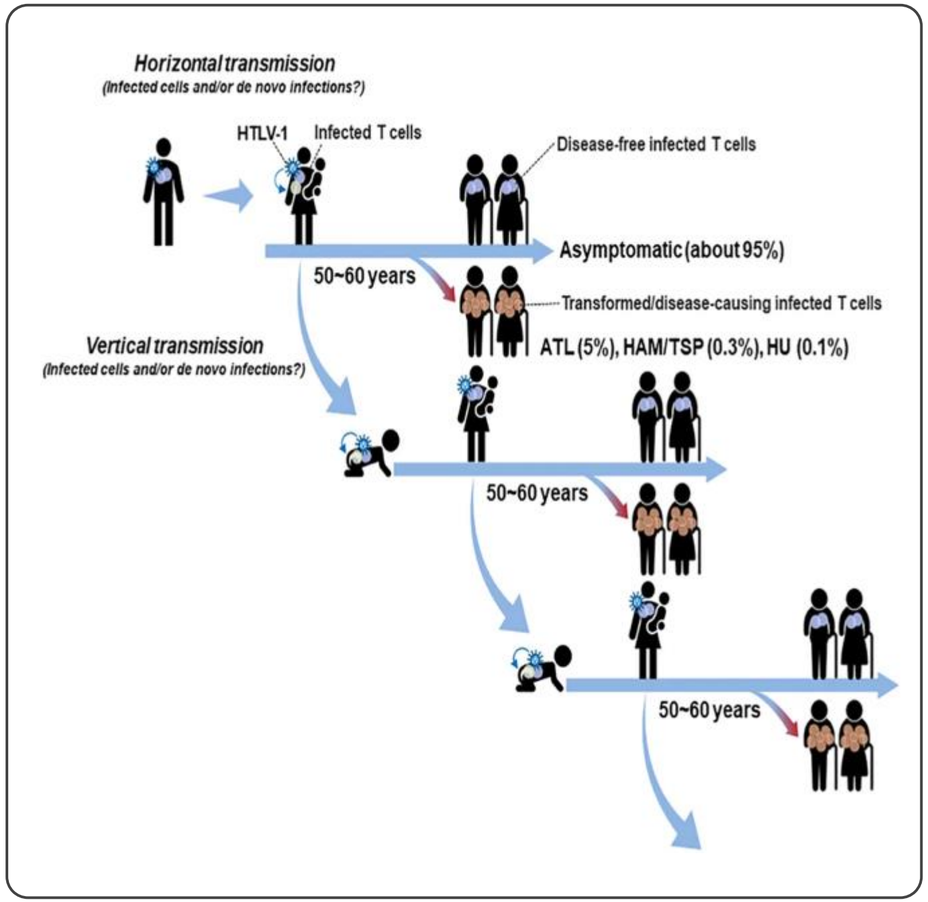
PMCID: PMC12821113 PMID: 41573066

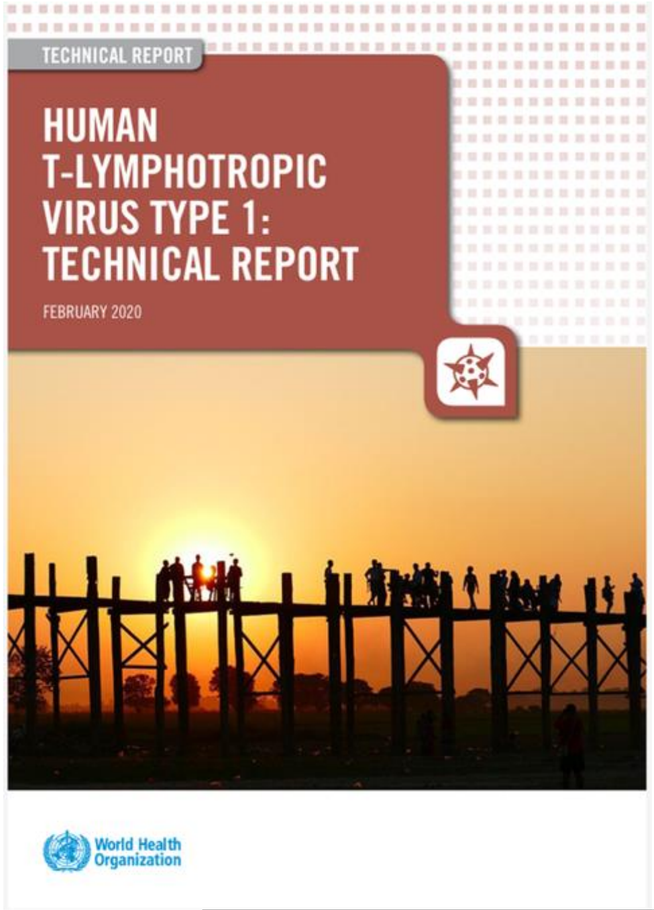
Turkiye and surrounding regions

Turkiye, strategically located between Europe, Asia, and the Middle East, has sparse data on HTLV prevalence. Limited studies among blood donors and high-risk populations have reported prevalence rates of less than 0.1% [17, 18]. However, the true burden in the general population remains unclear due to the lack of routine screening and epidemiological studies. Turkiye's close proximity to regions with higher HTLV prevalence, such as the Middle East and North Africa (MENA), raises concerns about potential under-detection [19]. This warrants further investigation, particularly in vulnerable groups such as immigrants, refugees, and rural populations.

- Erişkin T hücreli lösemi/lenfoma (ATL)
- HTLV-1 ilişkili miyelopati / tropikal spastik paraparesi (HAM/TSP)
- HTLV-1 ilişkili üveit
- İnfektif dermatit
- Bronşiolit / bronşiektazi
- Polimiyozit
- Artrit

- %95'i asemptomatik
- Bulaş vertikal (anne-çocuk) yada horizontal (cinsel yol/ kan transfüzyonu), Solid organ trans





Pharmaceutical intervention	Clinical trial phase	Recommended for clinical use	References
Interferon-based therapy			
Interferon-alpha + zidovudine	II	Yes	*(468–470)*
Interferon-alpha + arsenic	II	No (toxicity)	*(471)*
Interferon + zidovudine + arsenic	II	No (toxicity)	*(472)*
Chemotherapy			
Chemotherapy – various regimens	II–III	Yes Regimen specific	*(473–482)*
Chemotherapy (followed by interferon-alpha + zidovudine + lamivudine)	II	No	*(483)*
Biological agents			
Alemtuzumab	II	Insufficient evidence	*(484)*
Daclizumab	II	Insufficient evidence	*(485)*
Lenalidomide	II	Yes	*(486)*
Mogamulizumab	II	Yes	*(487)*
Mogamulizumab + chemotherapy	II	Yes	*(488,489)*

[a] Allogeneic hematopoietic stem cell transplant has not been evaluated in clinical trials among people with ATL, but it is a recommended management strategy in the appropriate clinical context.

TABLE 6. SUMMARY OF INTERVENTIONS EVALUATED IN CLINICAL TRIALS AMONG PEOPLE WITH HAM/TSP[a]

Pharmaceutical intervention	Clinical trial phase	Recommended for clinical use *(503)*
Agents postulated to alter disease course		
Corticosteroids Methylprednisolone (IV)	 Pilot, IV	 Yes
Cyclosporin	Pilot	Possible
Heparin	Pilot	No
HIV antiretroviral therapy Zidovudine + lamivudine Raltegravir Tenofovir disoproxil fumarate	 II Pilot Pilot	 No No No
Interferon-alpha and interferon-beta	Pilot, II, III	Consider as second-line therapy
Monoclonal antibody against interleukin-2 receptor (anti-Tac)	Pilot	No
Mogamulizumab	II	Insufficient evidence Ongoing evaluation
Sodium valproate	Pilot	No
Agents postulated to provide symptomatic benefit		
Danazol	II	No
Lactobacillus casei	Pilot	No
Pentosan polysulfate	Pilot	No
Pentoxifylline	Pilot	No
Prosultiamine	Pilot	No
Vitamin C	Pilot	No

[a] Other immunosuppressive or immunomodulatory agents that are used in clinical practice but have not been evaluated in clinical trials includ

News

Brazil takes a leap towards the elimination of HTLV-1 vertical transmission

Carolina Rosadas,[a,*] Draurio Barreira,[b] Pamela C. Gaspar,[b] Mayra G. Aragón,[b] Adijeane Oliveira,[c] Tatiane Assone,[d] and Angelica E. Miranda[b,**]

[a]Section of Virology, Department of Infectious Disease, Imperial College London, London, United Kingdom
[b]Brazilian Ministry of Health, Brasilia, Brazil
[c]HTLVida, Bahia, Brazil
[d]Faculty of Medicine, São Paulo University, São Paulo, Brazil

Human T-cell lymphotropic virus type 1 (HTLV-1) is associated with increased all-cause mortality and high morbidity and mortality diseases, including blood

There are, of course, several challenges ahead, particularly in a continental country such as Brazil.[7] Limited knowledge among healthcare professionals, and

The Lancet Regional Health - Americas
2024;39: 100888

Human T-lymphotropic virus type 1 and antiretroviral therapy: practical considerations for pre-exposure and post-exposure prophylaxis, transmission prevention, and mitigation of severe disease

Jake S O'Donnell, Noushin Jaberolansar, Keith J Chappell

Human T-lymphotropic virus type 1 (HTLV-1) is a retrovirus associated with substantial risk of secondary (often life-threatening) disease for the estimated 10 million to 20 million people infected globally. Despite a clear need, no HTLV-1-specific vaccine or antiretroviral therapy has been developed to date. Instead, existing public and primary health-care interventions inadequately focus on infection prevention and management of secondary diseases. In this Personal View, we discuss the evidence that exists to support the sensitivity of HTLV-1 to antiretroviral therapies approved by the US Food and Drug Administration for the treatment of HIV-1, how this sensitivity is affected by clinically relevant virological and immunological features, and additional practical considerations for the use of antiretroviral therapies in the context of HTLV-1.

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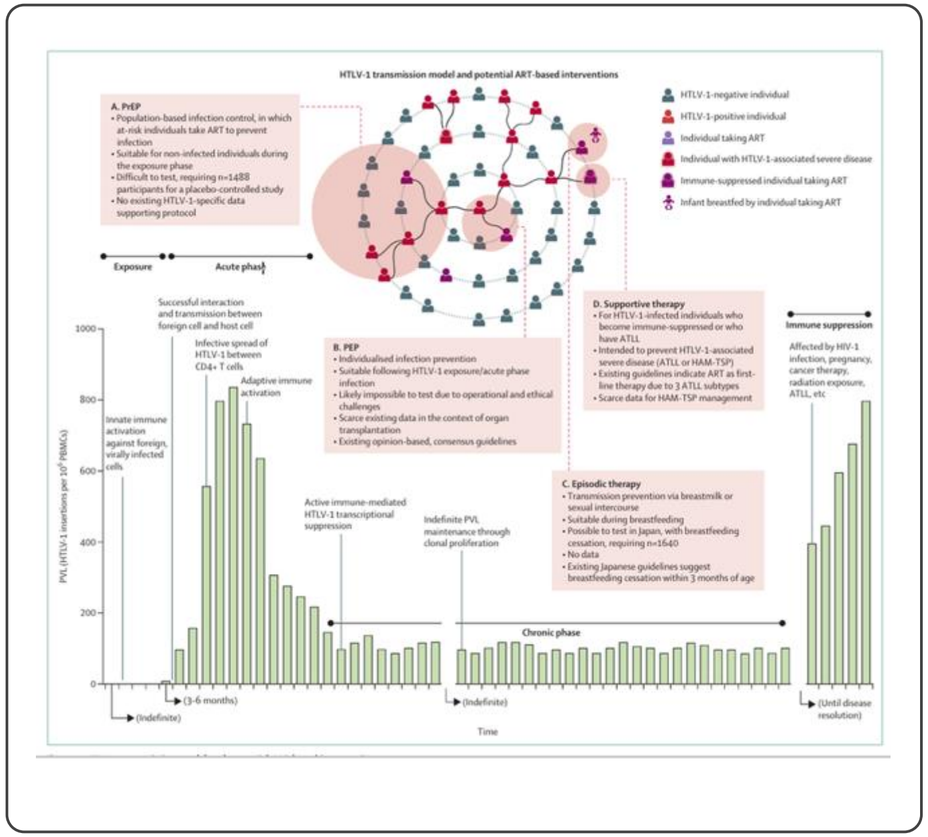
- **HTLV-1, ART'ye HIV-1'e göre daha az duyarlı ama yine de fayda olabilir**
- **Erken/profilaktik tedavi proviral yükü düşürebilir**
- **Bu azalma, uzun vadede hastalık riskini azaltabilir**



TEŞEKKÜRLER...