Prevention Of Dementia

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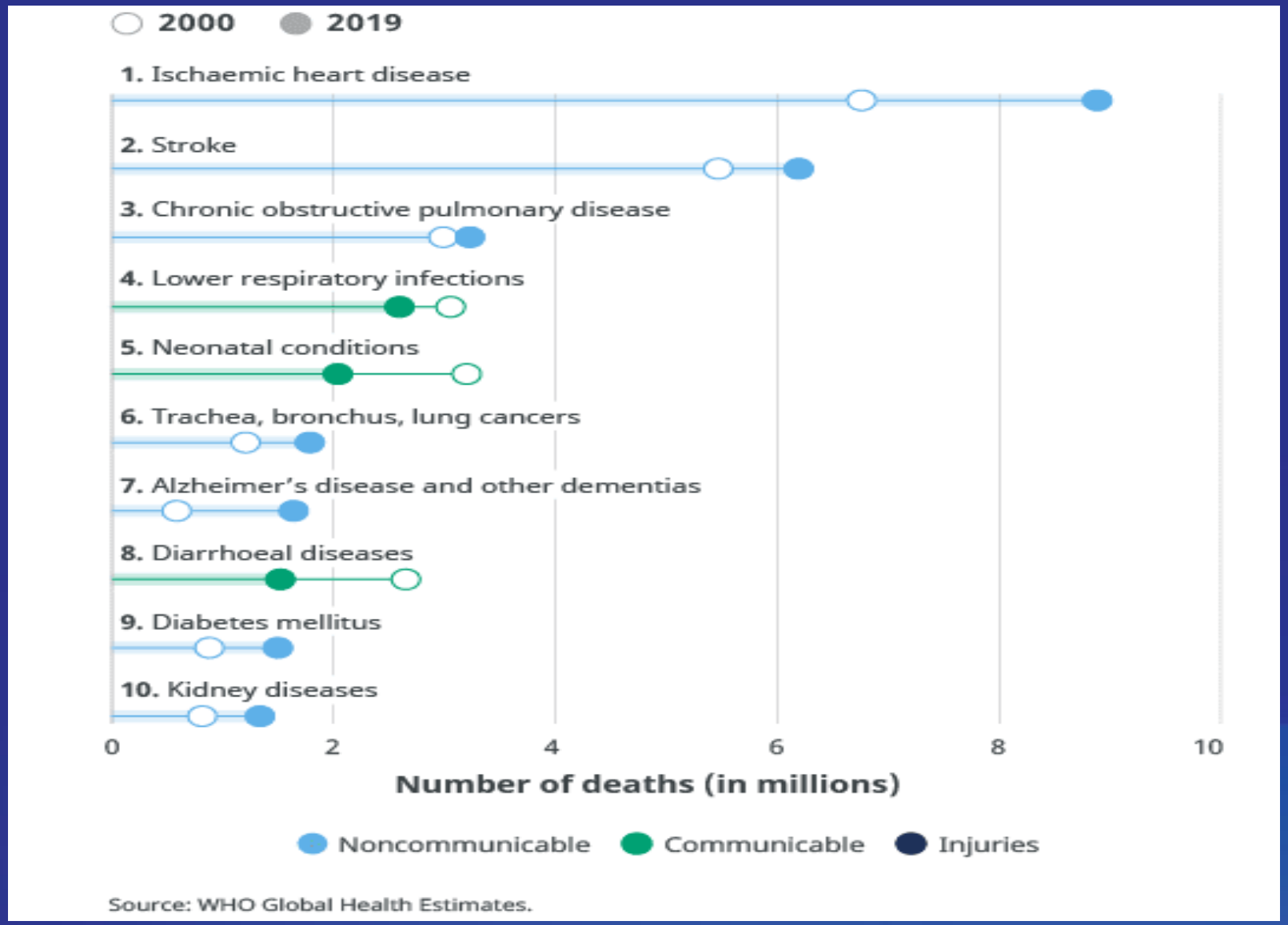
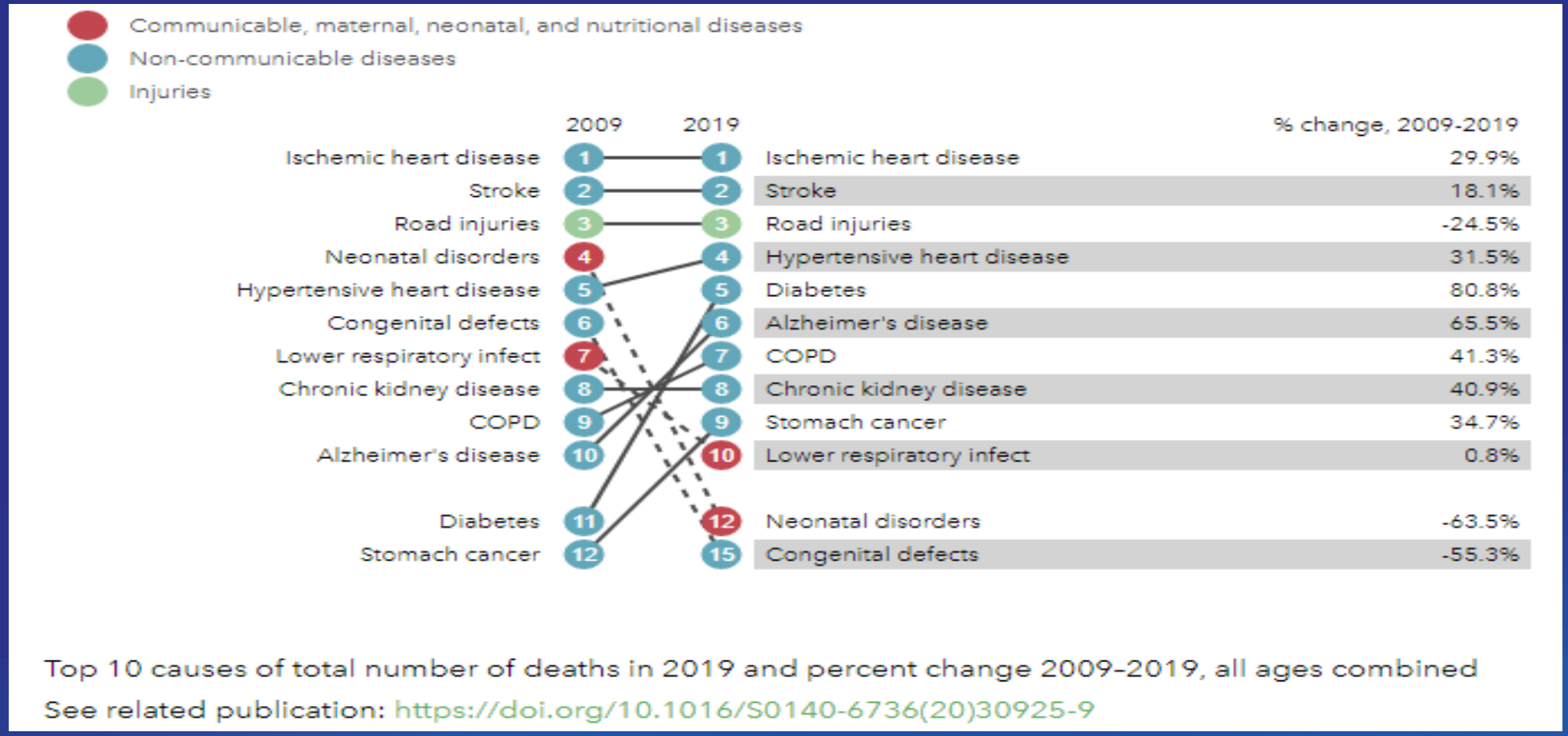
Leading causes of death globally



World Health Organization

https://www.who.int/news-room/fact-sheets/detail/the-top-10-causes-of-death

What causes the most deaths In Iran?



Top 10 causes of total number of deaths in 2019 and percent change 2009–2019, all ages combined

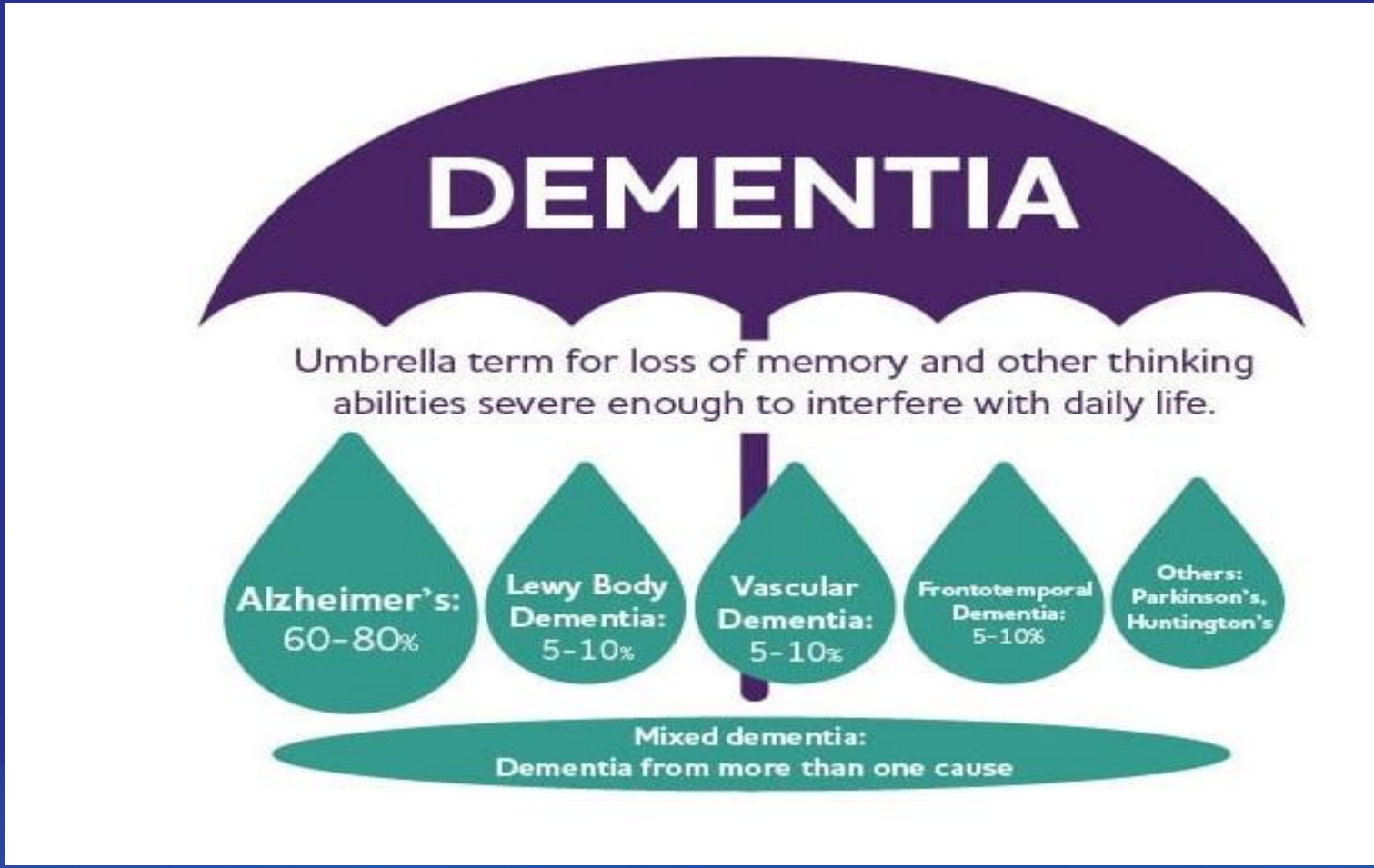
See related publication: https://doi.org/10.1016/S0140-6736(20)30925-9

http://www.healthdata.org/iran

Examples Of Neurodegenerative Diseases

- Involving the central nervous system:
  - Alzheimer's Disease
  - Parkinson Disease
  - ALS (Lou Gehrig's Disease)
  - Frontal Temporal Dementia
  - Huntington's Disease
  - Cerebellar Ataxias
  - Hereditary Spastic Paraplegias
- Involving the peripheral nervous system:
  - Charcot-Marie Tooth Hereditary Neuropathies
  - Muscular Dystrophy.

Examples Of Neurodegenerative Diseases



Dementia with Lewy bodies (DLB) is one of the most common causes of dementia after Alzheimer disease (AD) and vascular dementia.

DLB often presents a diagnostic challenge given this clinical heterogeneity and overlap with other neurodegenerative diseases.

DLB, although once considered rare, is recognized as a common cause of neurodegenerative dementia, affecting up to 5 percent of the general population and accounting for as much as 30 percent of all dementia cases.

Although genetic factors are an important component of DLB, the incidence of DLB is generally discordant among monozygotic twins , evidence that environmental or other epigenetic factors play an important role in the pathogenesis of DLB.

One study in community-based cohorts found that traumatic brain injury with loss of consciousness is associated with neocortical Lewy bodies in addition to PD and parkinsonism.

Probable DLB	■ Two or more core clinical features of DLB are present, with or without indicative biomarkers; **OR** ■ Only one core clinical feature is present, but with one or more indicative biomarkers ■ Probable DLB should not be diagnosed on the basis of biomarkers alone
Possible DLB	■ Only one core clinical feature of DLB is present, with no indicative biomarker evidence; **OR** ■ One or more indicative biomarkers are present, but there are no core clinical features

DLB is **less likely**	■ In the presence of any other physical illness or brain disorder including cerebrovascular disease, sufficient to account in part or in total for the clinical picture* ■ If parkinsonian features are the only core clinical feature and appear for the first time at a stage of severe dementia ¶

Visual hallucinations occur in up to 70 percent of patients with DLB; they are an early sign in DLB and may precede parkinsonism.

Tremor is less common and less severe than in PD.

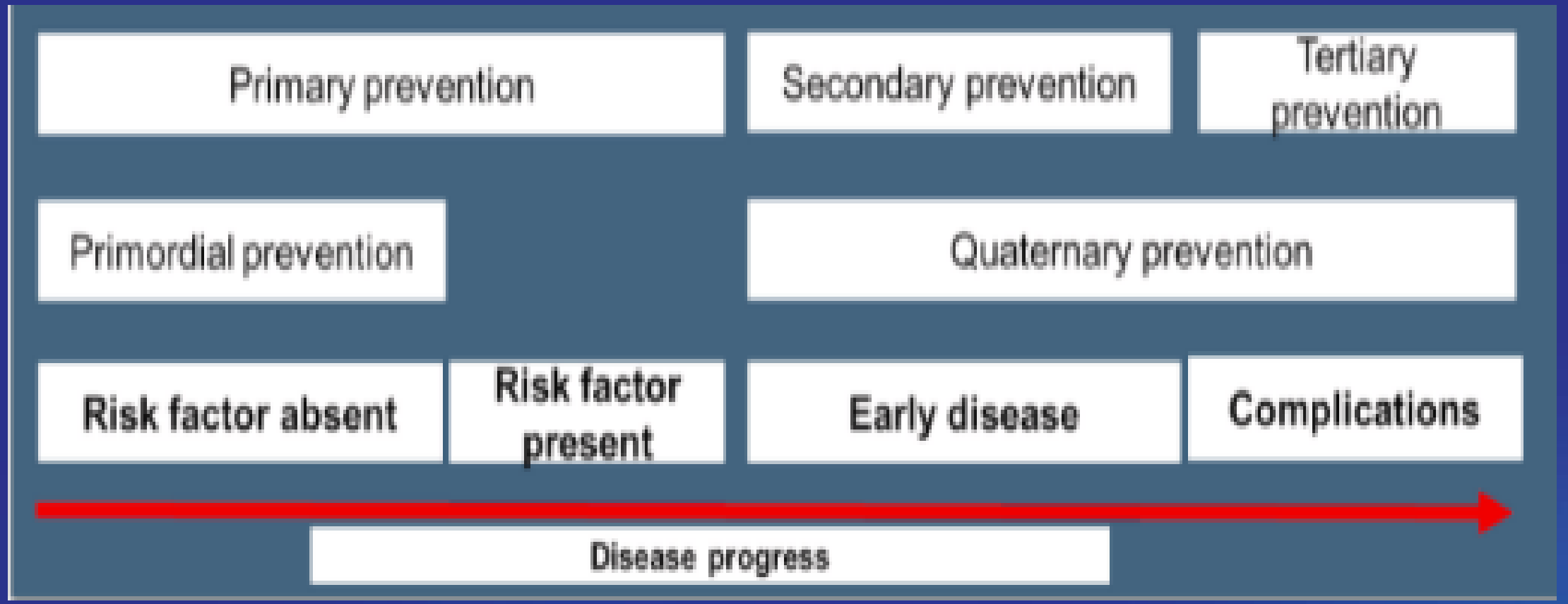
Approximately 30 to 50 percent of individuals with DLB have marked sensitivity to antipsychotic drugs .

Acute reactions include severe, sometimes irreversible parkinsonism and impaired consciousness, sometimes with other features suggestive of neuroleptic malignant syndrome. This can occur in individuals without baseline parkinsonism.

Adverse reactions are more common with first-generation antipsychotics (eg, haloperidol), but reactions to second-generation antipsychotics have also been described.

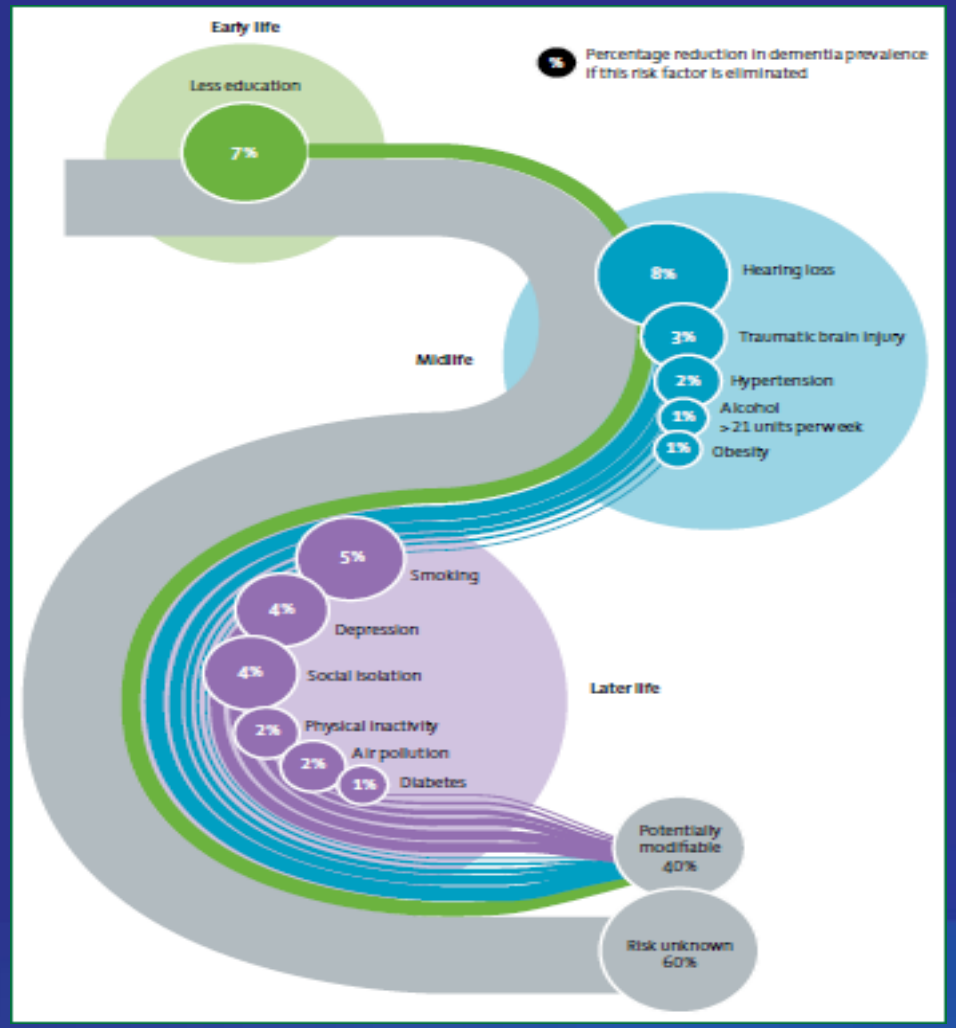
Recurrent falls occur in up to one-third of patients with DLB and may be among the earliest symptoms.

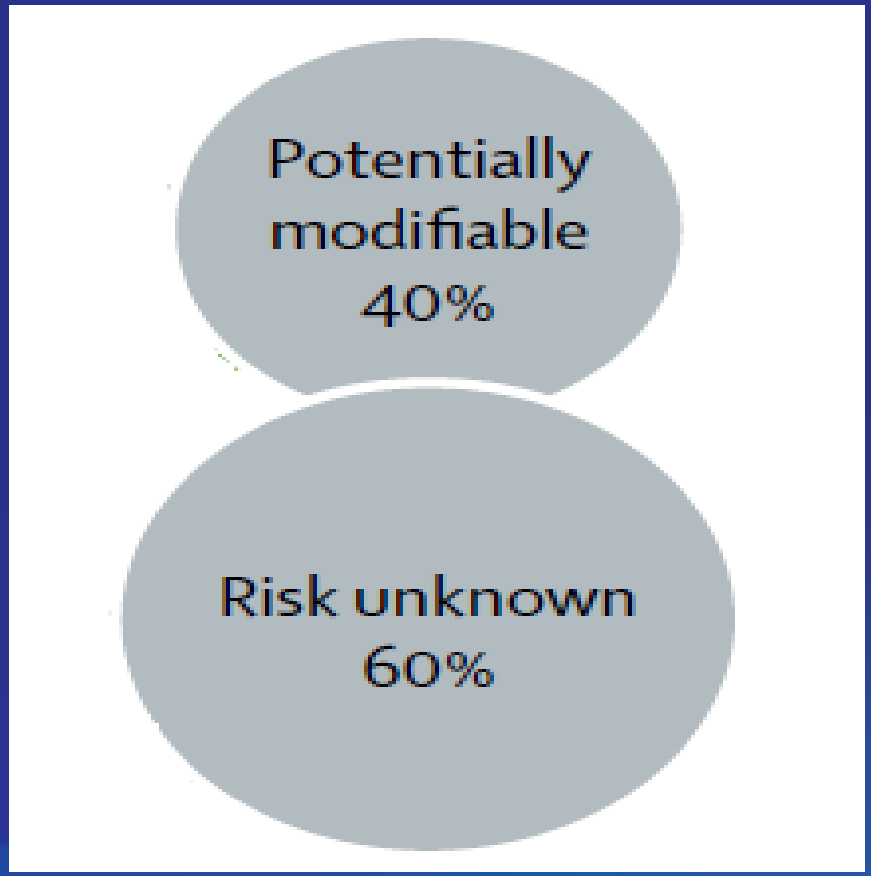
Falls may occur with or without provocation and may be related to parkinsonism, to cognitive fluctuations, or to orthostatic hypotension.


Primary prevention
Secondary prevention
Tertiary prevention
Primordial prevention
Quaternary prevention
Risk factor absent
Risk factor present
Early disease
Complications
Disease progress

Type/level of prevention	Aim of measures
Primordial	Control of single risk factors
Primary	Prevention of disease
Secondary (early)	Identification of the disease in its early asymptomatic stage
Tertiary (late)	Prevention of disease complications
Quaternary	Prevention of unfounded or harmful medical activities

Modifiable risk factors for dementia





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Modifiable risk factors for dementia

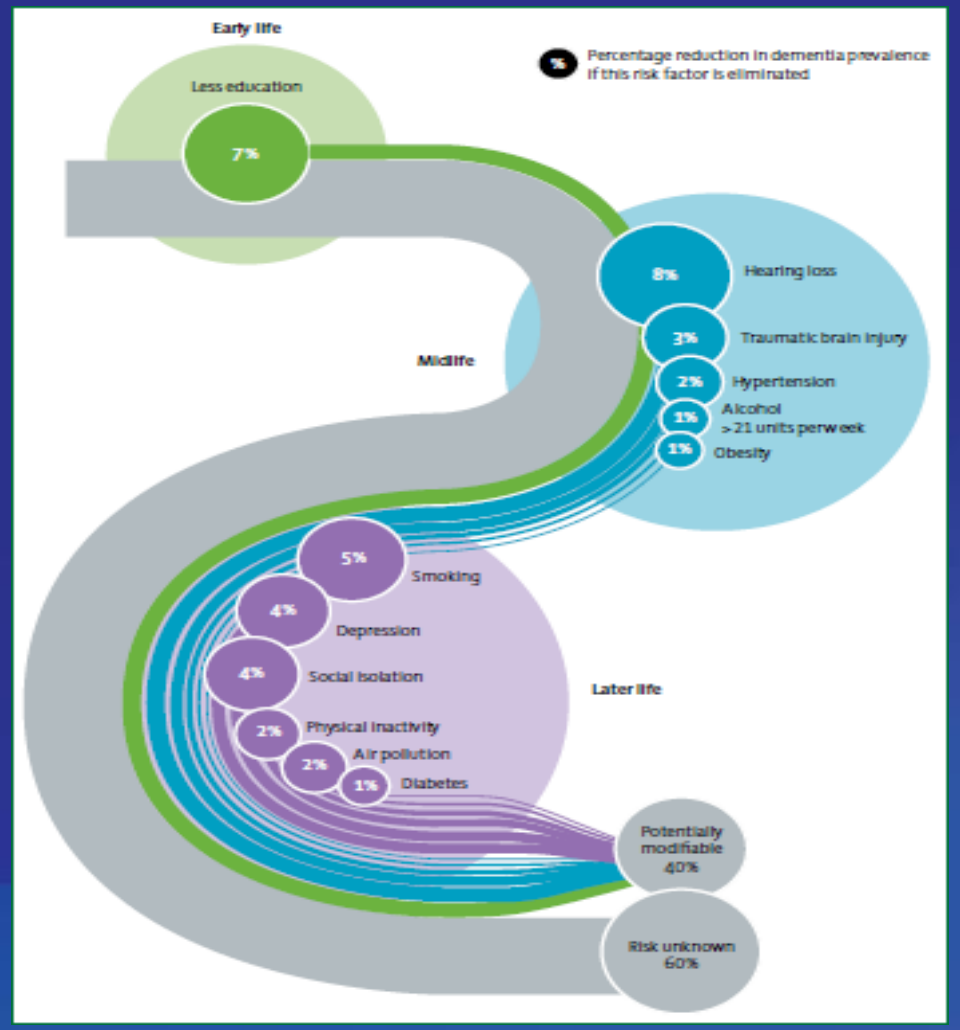
The age-specific incidence of dementia has fallen in many countries.

less education
Hypertension
Hearing impairment
Smoking
Obesity
Depression
Physical inactivity
Diabetes
low social contact

Excessive alcohol consumption

Traumatic brain injury (TBI)

Air pollution



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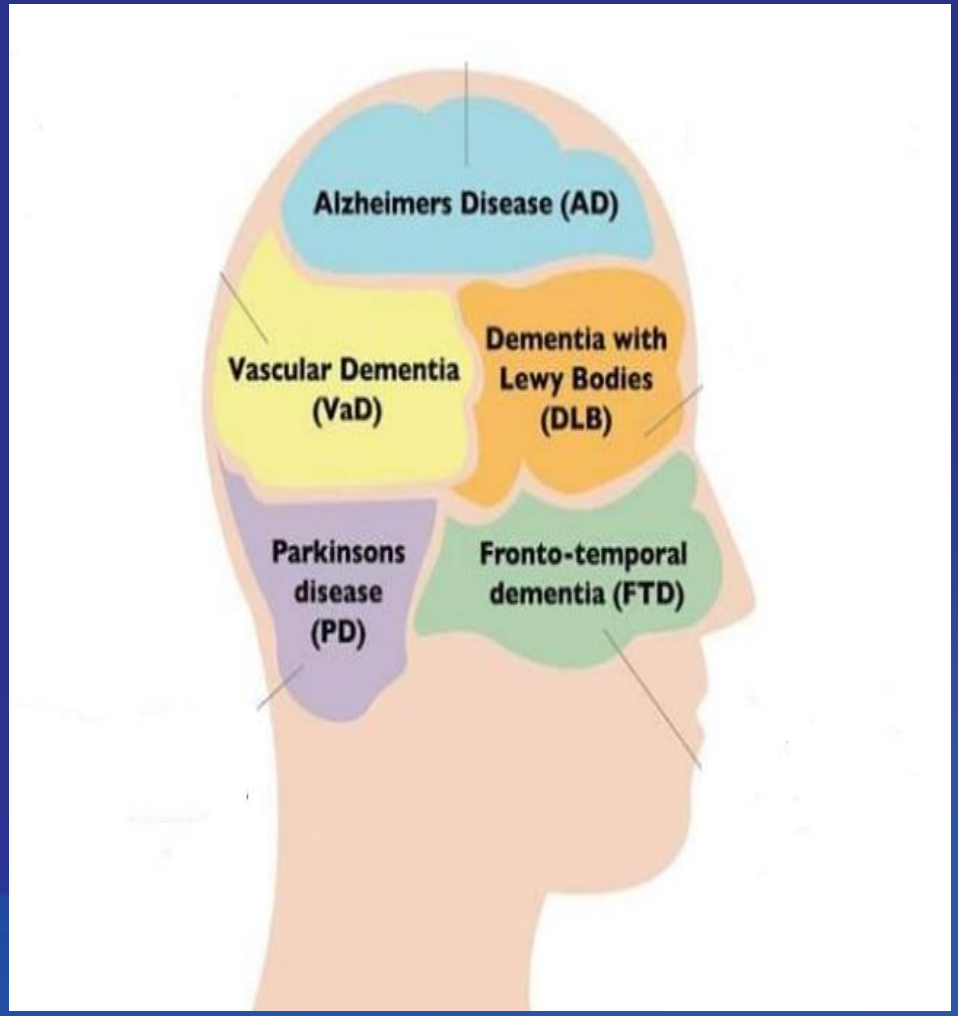
Modifiable risk factors for dementia

It is never too early and never too late in the life course for dementia prevention.

Early-life (younger than 45 years) risks, such as less education, affect cognitive reserve.

Midlife (45–65 years), and later-life (older than 65 years) risk factors influence reserve and triggering of neuropathological developments.



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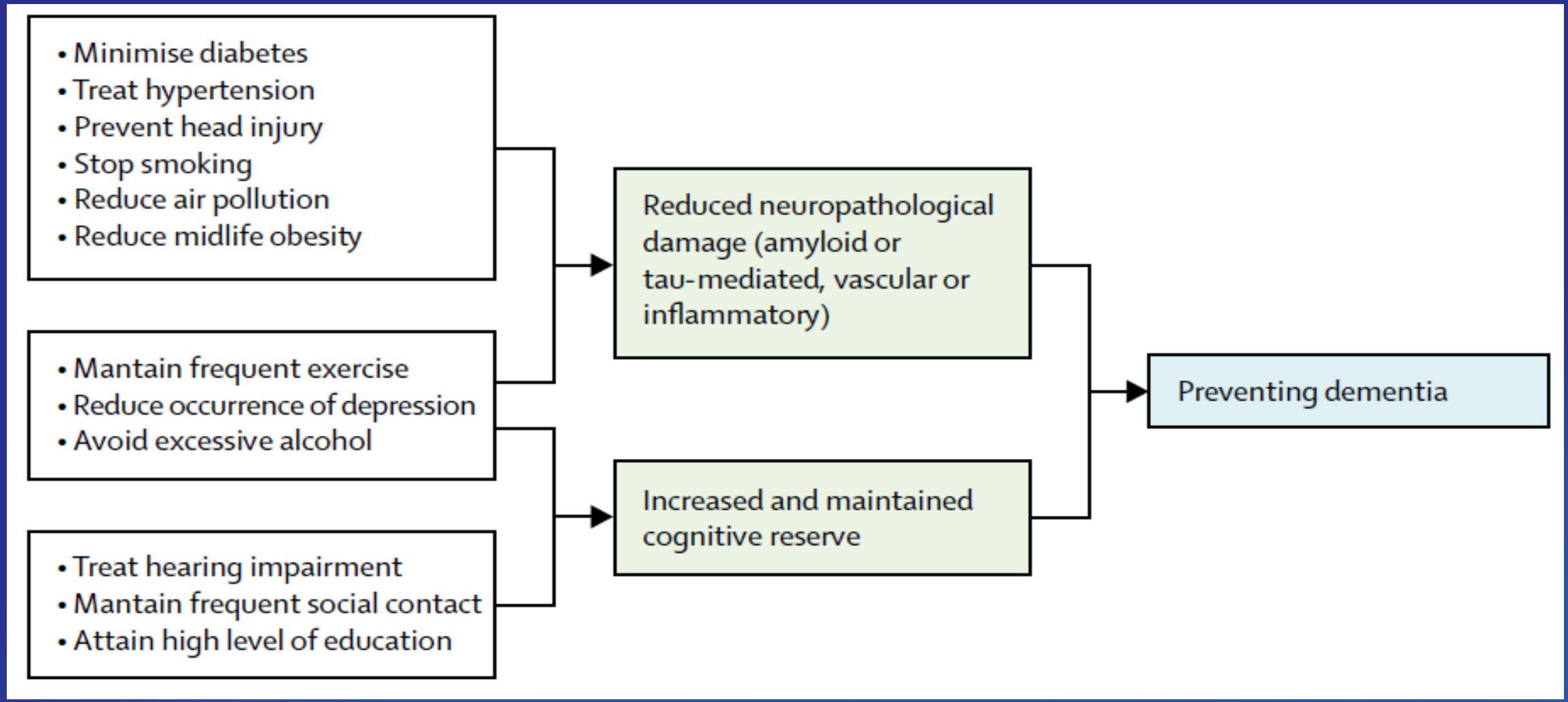
Modifiable risk factors for dementia



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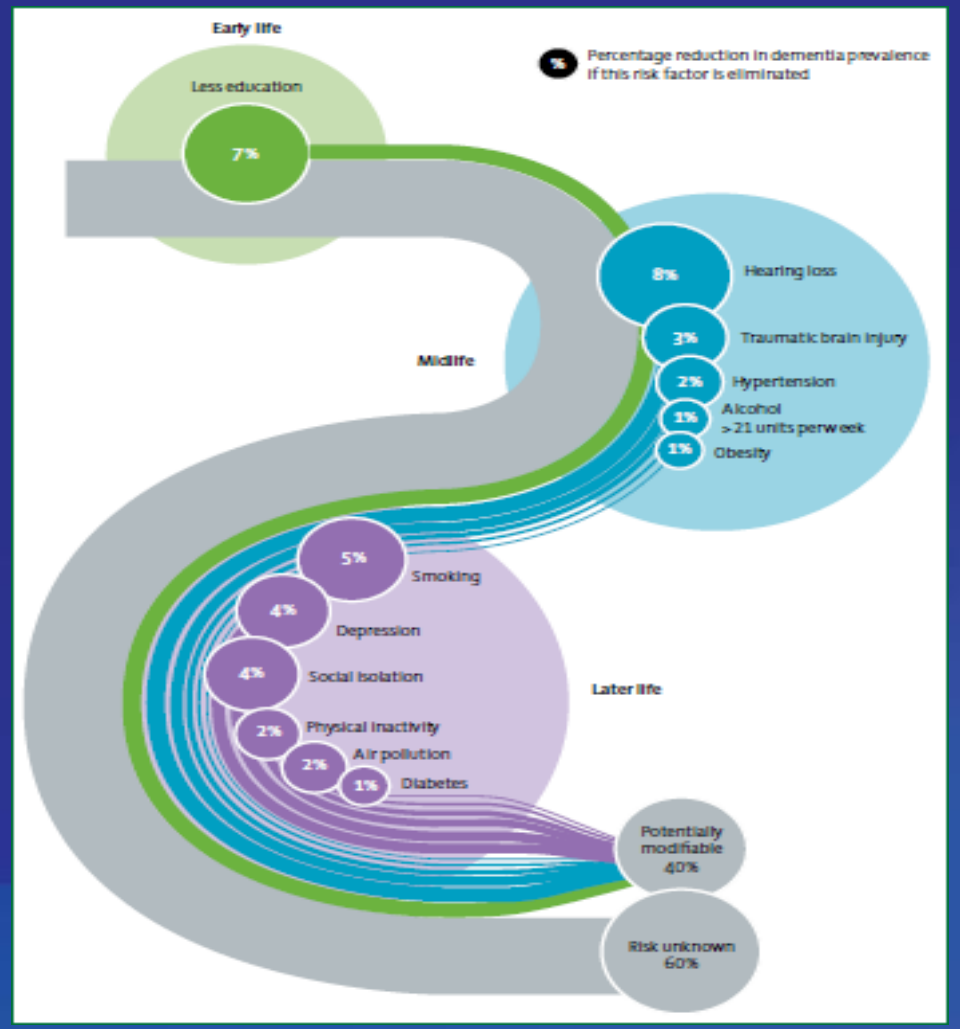
Modifiable risk factors for dementia

	Relative risk for dementia (95% CI)	Risk factor prevalence	Communality	Unweighted PAF	Weighted PAF*
Early life (<45 years)					
Less education	1·6 (1·3–2·0)	40·0%	61·2%	19·4%	7·1%
Midlife (age 45–65 years)					
Hearing loss	1·9 (1·4–2·7)	31·7%	45·6%	22·2%	8·2%
TBI	1·8 (1·5–2·2)	12·1%	55·2%	9·2%	3·4%
Hypertension	1·6 (1·2–2·2)	8·9%	68·3%	5·1%	1·9%
Alcohol (>21 units/week)	1·2 (1·1–1·3)	11·8%	73·3%	2·1%	0·8%
Obesity (body-mass index ≥30)	1·6 (1·3–1·9)	3·4%	58·5%	2·0%	0·7%
Later life (age >65 years)					
Smoking	1·6 (1·2–2·2)	27·4%	62·3%	14·1%	5·2%
Depression	1·9 (1·6–2·3)	13·2%	69·8%	10·6%	3·9%
Social isolation	1·6 (1·3–1·9)	11·0%	28·1%	4·2%	3·5%
Physical inactivity	1·4 (1·2–1·7)	17·7%	55·2%	9·6%	1·6%
Diabetes	1·5 (1·3–1·8)	6·4%	71·4%	3·1%	1·1%
Air pollution	1·1 (1·1–1·1)	75·0%	13·3%	6·3%	2·3%

Data are relative risk (95% CI) or %. Overall weighted PAF=39·7%. PAF=population attributable fraction. TBI=traumatic brain injury. *Weighted PAF is the relative contribution of each risk factor to the overall PAF when adjusted for communality.

Table 1: **PAF for 12 dementia risk factors**



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Education and midlife and late-life cognitive stimulation

Higher childhood education levels and lifelong higher educational attainment reduce dementia risk.

late adolescence

After age 20 years

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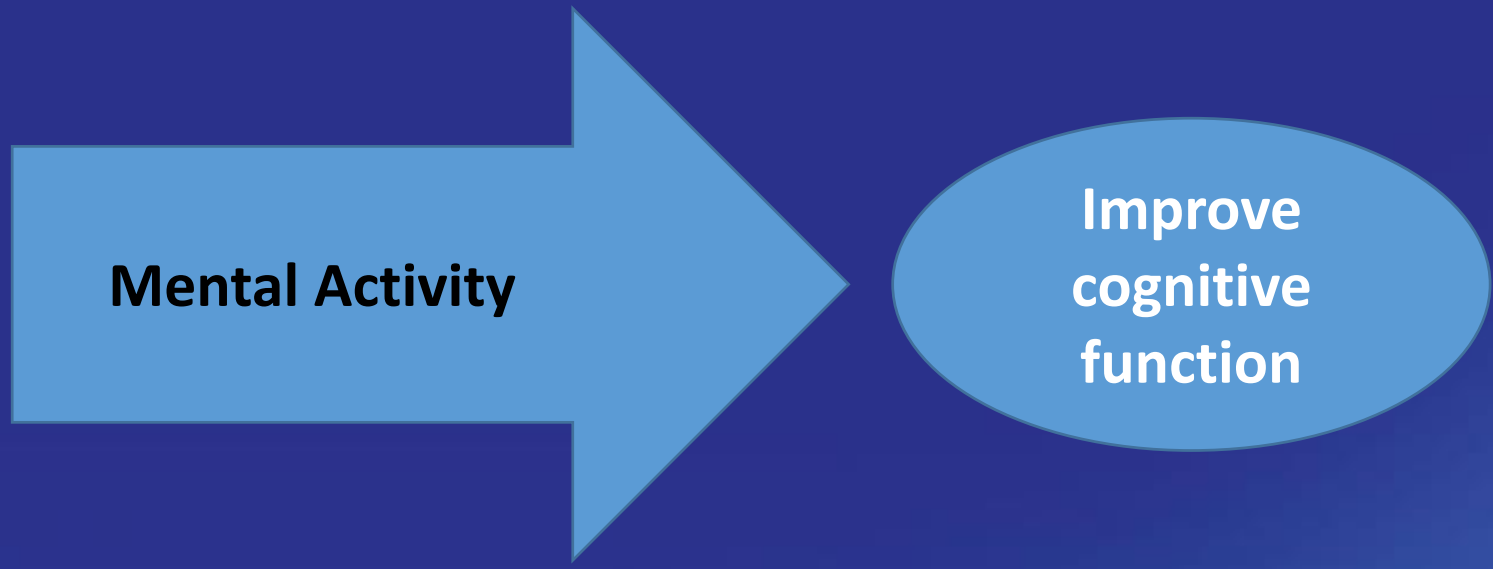
Education and midlife and late-life cognitive stimulation

People in more cognitively demanding jobs tend to show less cognitive deterioration before, and sometimes after retirement than those in less demanding jobs.



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Education and midlife and late-life cognitive stimulation

Cognitive interventions in normal cognition and mild cognitive impairment ??????

Prevent Dementia

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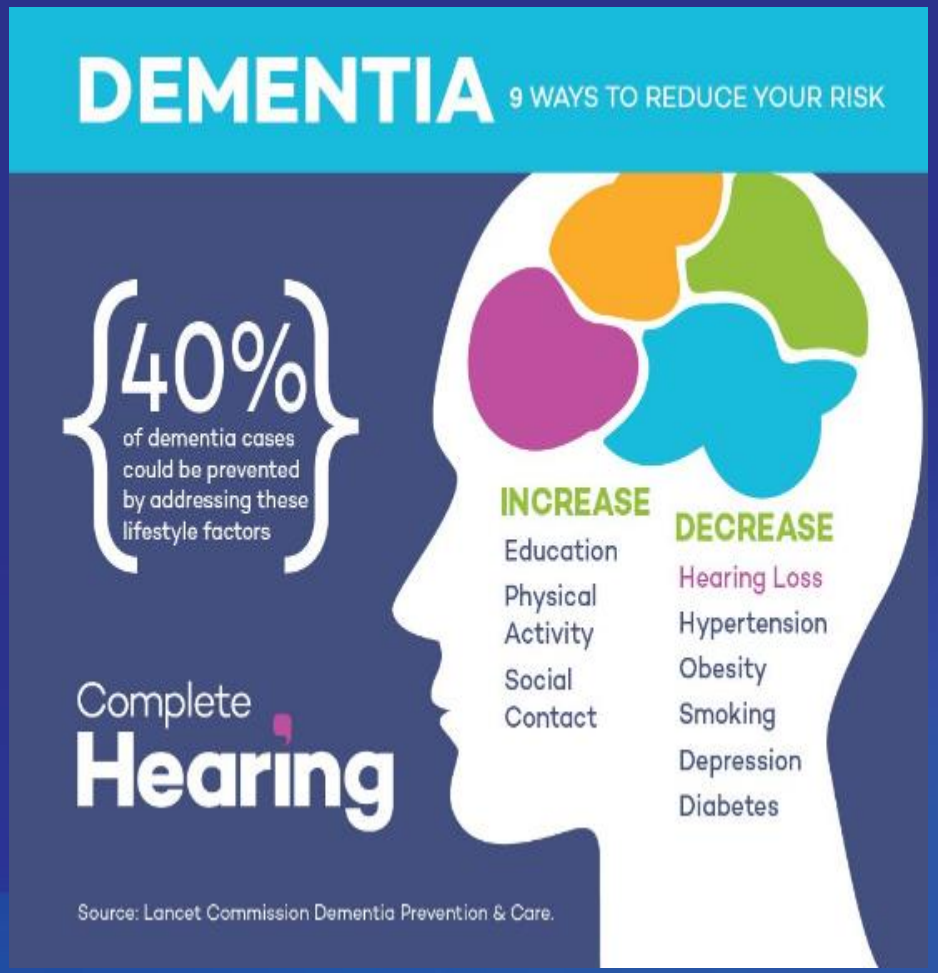
Hearing impairment

A cross-sectional study of 6451 individuals designed to be representative of the US population, with a mean age of 59·4 years, found a decrease in cognition with every 10 dB reduction in hearing, which continued to below the clinical threshold so that subclinical levels of hearing impairment (below 25 dB) were significantly related to lower cognition.



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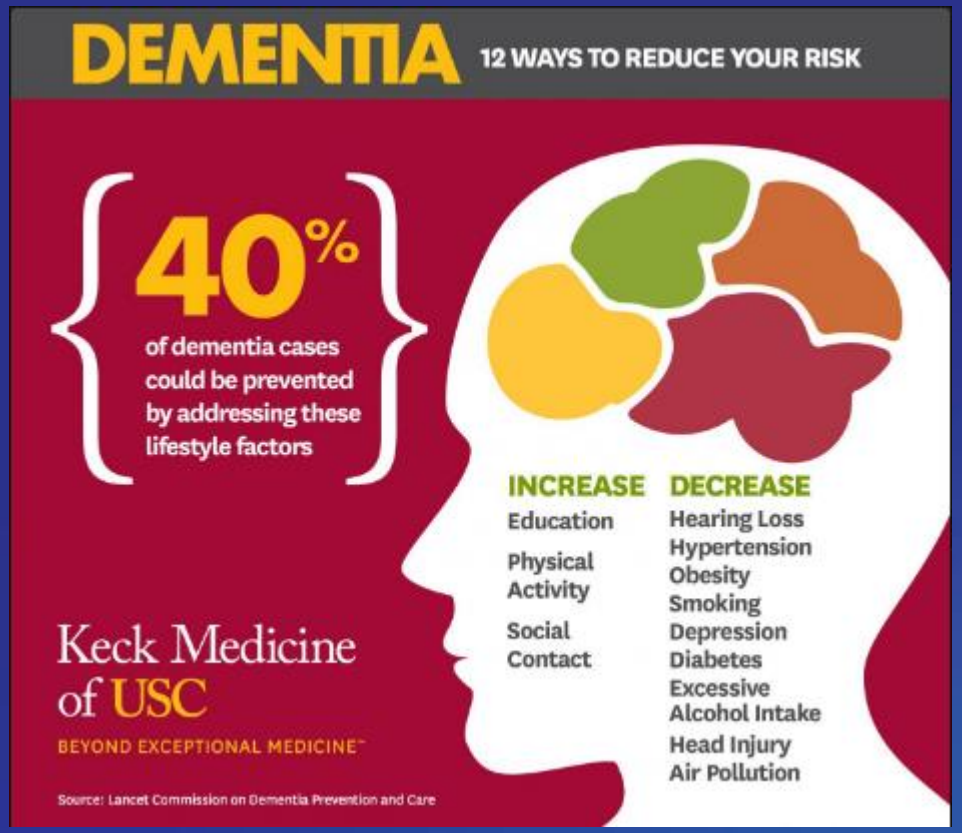
Hearing impairment

A 25-year prospective study of 3777 people aged 65 years or older found increased dementia incidence in those with self-reported hearing problems except in those using hearing aids.



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Hearing impairment

Hearing aid use was the largest factor protecting from decline (regression coefficient β for higher episodic memory 1 · 53; p<0 · 001) adjusting for protective and harmful factors.

The long follow-up times in these prospective studies suggest hearing aid use is protective, rather than the possibility that those developing dementia are less likely to use hearing aids.

Hearing loss might result in cognitive decline through reduced cognitive stimulation.

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Traumatic brain injury (TBI)

The International Classification of Disease (ICD) defines **mild TBI** as concussion and **severe TBI** as skull fracture, oedema, brain injury or bleed.

Dementia risk was highest in the 6 months after TBI and increased with number of injuries in people with TBI .

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Hypertension

Persistent midlife hypertension is associated with increased risk of a late life dementia.

In the Framingham Offspring cohort comprising 1440 people, **elevated systolic blood pressure (≥140 mm Hg in midlife; mean age 55 years)** was associated with an increased risk of developing dementia over an 18 year follow-up period.

In the same cohort, people in **late midlife** (mean age 62 years) with ideal cardiovascular parameters (current non-smoker, body mass index [BMI] 18 · 5–25 kg/m2, regular physical activity, healthy diet, optimum blood pressure <120/<80 mm Hg, cholesterol, and normal fasting blood glucose) were compared to people with at least one of these risks. Those with ideal cardiovascular parameters had a lower 10-year risk of all-cause dementia vascular dementia and clinically diagnosed Alzheimer's disease.

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Physical inactivity, exercise, and fitness

Meta-analyses of longitudinal observational studies of 1–21 years duration showed exercise to be associated with reduced risk of dementia.

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Diabetes

Diabetes as a risk factor for dementia.

Distinguishing between treated and untreated diabetes as a risk factor for dementia is challenging .

The effect of different diabetic medications on cognition or dementia outcomes remains unclear.

People with diabetes taking metformin had lower prevalence of cognitive impairment compared with those taking other medications or no medication.

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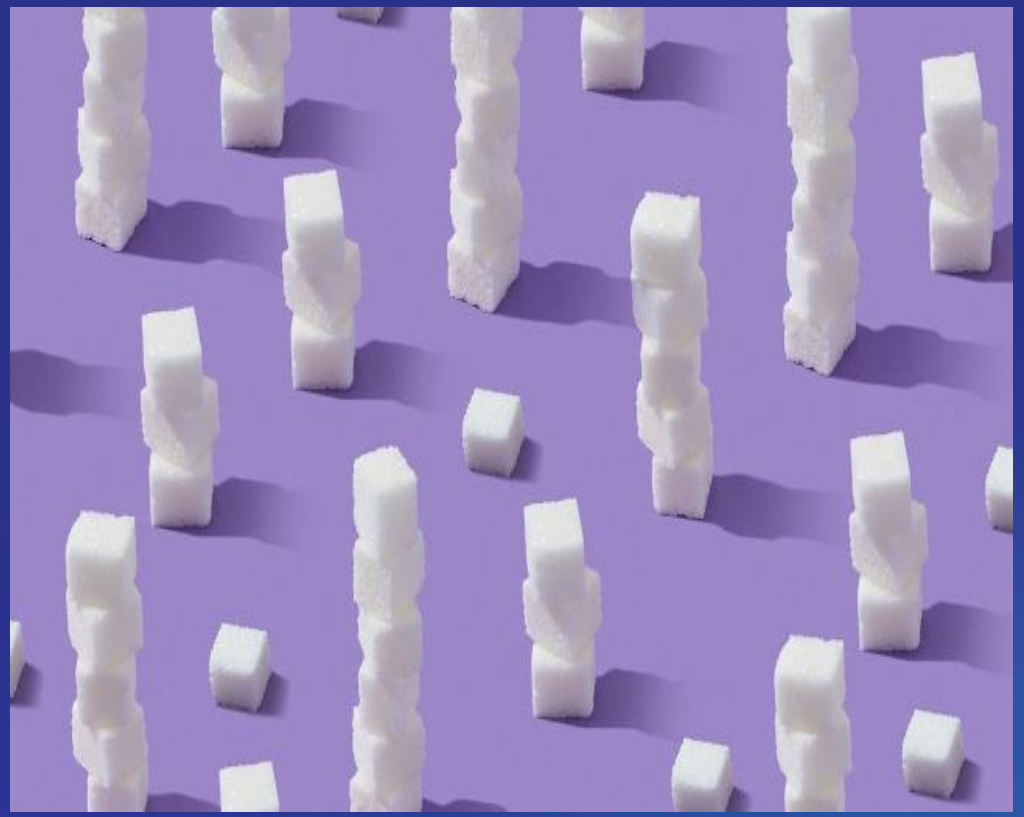
Diabetes

Overall type 2 diabetes is a clear risk factor for development of future dementia; however, whether any particular medication ameliorates this risk is unclear.

Intensive diabetic control does not decrease the risk of dementia.



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Excessive alcohol consumption

Heavy drinking is associated with brain changes, cognitive impairment, and dementia, a risk known for centuries.

The relationship of dementia with alcohol use disorders was particularly clear in the earlier onset dementias (age less than 65 years) in which 56 · 6% had an alcohol use disorder noted in their records.

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Weight control and obesity

Overweight is an emerging concern, given the changing BMI across the world's ageing population.

New evidence supports the relationship between increased BMI and dementia .People aged 35 to 65 years, followed up for up to 42 years.
It reported obesity (BMI ≥30) but not being overweight (BMI 25–30) was associated with late-life dementia.

Higher body mass measured before probable preclinical and prodromal dementia was associated with increased dementia .

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Smoking

Smokers are at higher risk of dementia than nonsmokers, 2 and at a higher risk of premature death before the age at which they might have developed dementia, introducing some bias and uncertainty in the association between smoking and risk of dementia.

Stopping smoking, even when older, reduces this risk.

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Smoking

35% of nonsmoking adults and 40% of children are estimated to be exposed to second-hand smoke; although literature on the impact of this exposure and dementia risk is scarce.

One study indicated that in women aged 55–64 years, second-hand smoke exposure was associated with more memory deterioration and the risk increased with exposure duration even after controlling for other confounding factors.

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Depression

Depression is associated with dementia incidence, with a variety of possible psychological or physiological mechanisms.

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Social Contact

Social contact, now an accepted protective factor, enhances cognitive reserve or encourages beneficial behaviours, although isolation might also occur as part of the dementia prodrome.

less social contact increases the risk of dementia.

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Air pollutants

Animal models suggest airborne particulate pollutants accelerate neurodegenerative processes through cerebrovascular and cardiovascular disease, Aβ deposition, and amyloid precursor protein processing.

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Sleep

Mechanisms by which sleep might affect dementia remain unclear, but sleep disturbance has been linked with β-amyloid (Aβ) deposition, reduced glymphatic clearance pathways activation, low grade inflammation, increased Tau, hypoxia and cardiovascular disease.

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Diet

Nutrition and dietary components are challenging to research with controversies still raging around the role of many micronutrients and health outcomes in dementia.

There has been a move towards considering the evidence base for whole diets in the last 5 years, particularly high plant intake such as in the Mediterranean diet (high intake of vegetables, legumes, fruits, nuts, cereals, and olive oil; low intake of saturated lipids and meat) or the similar Nordic diet, rather than individual nutrients, which might reduce cognitive decline and dementia.

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Panel: Recommended strategies for dementia risk reduction

Risks are particularly high in more socially disadvantaged populations including in Black, Asian, and minority ethnic groups.

Population-wide

- Prioritise childhood education for all, worldwide
- Implement social public health policies that reduce hypertension risk in the entire population
- Develop policies that encourage social, cognitive, and physical activity across the life course for all (with no evidence for any specific activities being more protective)
- Scrutinise the risks for hearing loss throughout the life course, to reduce the risk of exposure to this risk factor
- Reduce the risk of serious brain trauma in relevant settings, including occupational and transport
- National and international policies to reduce population exposure to air pollution
- Continue to strengthen national and international efforts to reduce exposure to smoking, both for children and adults, and to reduce uptake and encourage cessation

Targeted on individuals

- Treat hypertension and aim for systolic blood pressure <130 mm Hg in midlife
- Use hearing aids for hearing loss; we need to help people wear hearing aids as many find them unacceptable, too difficult to use, or ineffective
- Avoid or discourage drinking 21 or more units of alcohol per week
- Prevent head trauma where an individual is at high risk
- Stopping smoking is beneficial regardless of age
- Reduce obesity and the linked condition of diabetes by healthy food availability and an environment to increase movement
- Sustain midlife, and possibly late-life physical activity

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دانشگاه علوم پزشکی و خدمات بهداشتی درمانی تهران

برنامه کشوری دمانس در نظام سلامت ایران
جهت اجرا در بازه زمانی ۱۴۰۵-۱۴۰۰

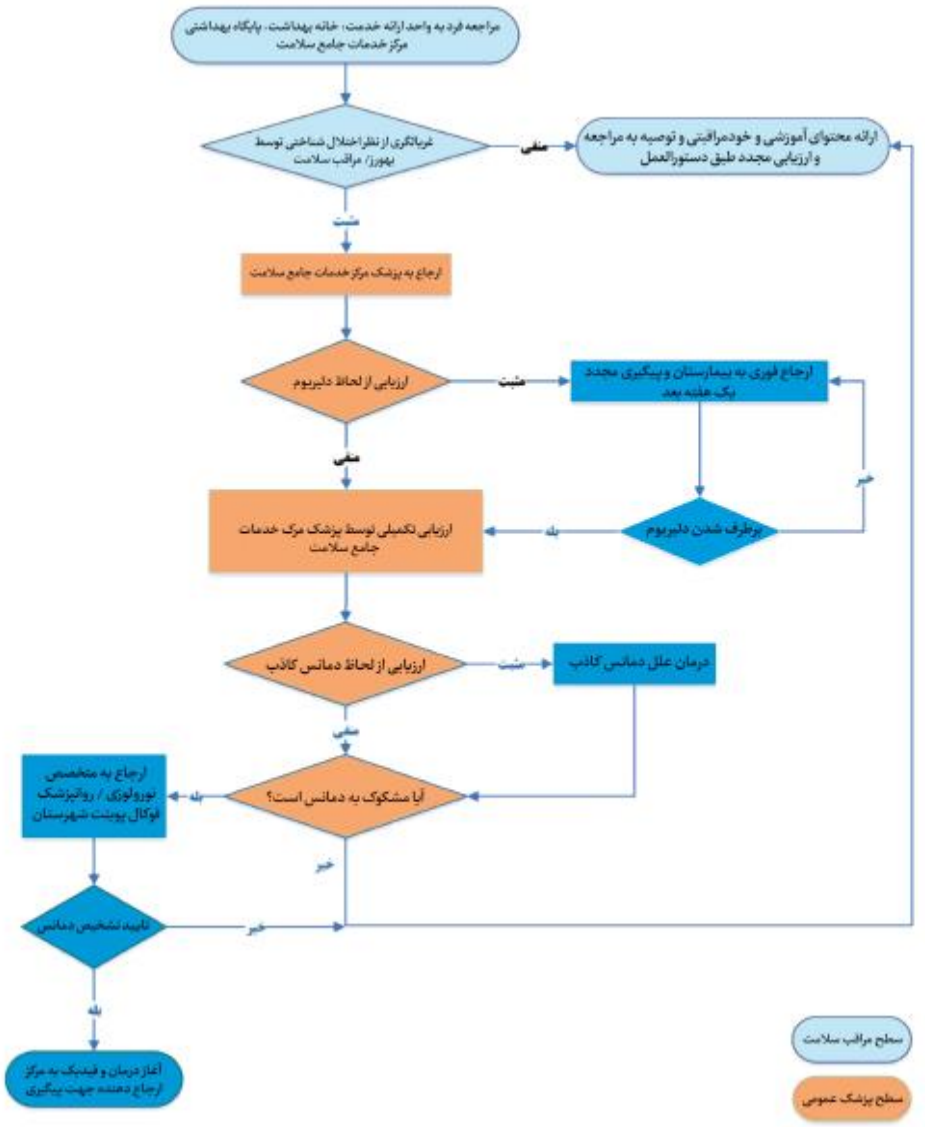
معاونت بهداشت وزارت بهداشت، درمان و آموزش پزشکی:
دفتر سلامت روانی، اجتماعی و اعتیاد
دفتر سلامت جمعیت، خانواده و مدارس

معاونت درمان وزارت بهداشت، درمان و آموزش پزشکی:
مرکز مدیریت پیوند و درمان بیماری ها
دفتر مشاور وزیر در امور توانبخشی

خرداد ۱۴۰۰
ناشر: اندیشه ماندگار
نوبت چاپ: اول - ۱۴۰۰
شابک: ۹۷۸-۶۲۲-۷۰۰۷-۶۵-۷

انتشارات اندیشه ماندگار
قم - خیابان صفائیه (شهدا) - انتهای کوی بیگدلی - نبش کوی شهید گلدوست - پلاک ۲۸۴
تلفن: ۰۲۵/۳۷۷۴۲۱۴۲-۳۷۷۴۶۱۶۵ همراه: ۰۹۱۲۲۵۲۶۲۱۳

فلوچارت اصلی فرایند غربالگری و تشخیص و درمان دمانس در نظام سلامت



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