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## Women's excess unhealthy life years: disentangling the unhealthy life years gap

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**Background:** Compared to men, women live longer but have more years with disability. We assessed the contribution of gender differences in mortality and disability, total and by cause, to women's excess unhealthy life years (ULYs). **Methods:** We used mortality data for France 2008 from Eurostat, causes of death from the CépiDc-INSERM-database; and disability and chronic conditions data from the French Disability Health Survey 2008–09. ULYs were calculated by the Sullivan method. The contributions of mortality and disability differences to gender differences in ULY were based on decomposition analyses. **Results:** Life expectancy of French women aged 50 was 36.3 years of which 19.0 were ULYs; life expectancy of men was 30.4 years of which 14.2 were ULYs. Of the 4.8 excess ULYs in women, 4.0 years were due to lower mortality. Of these 4.0 ULYs, 1.8 ULY originated from women's lower mortality from cancer, 0.8 ULY from heart disease and 0.3 ULY from accidents. The remaining 0.8 excess ULY in women were from higher disability prevalence, including higher disability from musculoskeletal diseases (+1.8 ULY) and anxiety-depression (+0.6 ULY) partly offset by lower disability from heart diseases (–0.8 ULY) and accidents (–0.3 ULY). **Conclusion:** Lower mortality and higher disability prevalence contributed to women's longer life expectancy with disability. Women's higher disability prevalence due to non-fatal disabling conditions was partly offset by lower disability from heart disease and accidents. Conditions differentially impact gender differences in ULY, depending on whether they are mainly life-threatening or disabling. The conclusions confirm the health-survival paradox.

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## Introduction

Women experience lower mortality, but report more disability compared to men of the same age. The female longevity advantage and the 'health-survival paradox' have received much attention in the scientific literature, but the fact that this implies that women live many more years with disability has received scant attention.<sup>1–4</sup> In general, the focus of past research has been on total life expectancy and on healthy life expectancy, in Europe measured with the healthy life years (HLYs).<sup>5</sup> Gender differences in HLY are generally much smaller than in life expectancy owing to opposite gender differences in mortality and disability which reduce the gap in HLY. In some countries, women have fewer HLYs than men indicating that their mortality advantage is offset by their disability disadvantage. However, the same opposing gender differences increase the gender gap in unhealthy life years (ULYs)<sup>2,3,6</sup> and gender differences in ULY may even be larger than gender differences in life expectancy.<sup>3</sup>

Insight into the gender gap in ULY and the role of differences in mortality and disability, both in general and from specific chronic conditions, is relevant to better understand gender disparities and to optimize strategies to reduce these inequalities. A high contribution of a specific disease may point to specific risk factors, possibly modifiable.

The aim of this study is to improve understanding of gender disparities in ULY by quantifying the contribution of mortality and disability differences to the origin of gender gap in ULYs, both in total and from different causes of death and disability. We will address the following questions: (i) what is the contribution of women's lower mortality (extending the time at risk of disability) and higher prevalence of disability to the gender gap in ULY and (ii) which causes of death and disability contribute most to this gender difference. We focus on France, given the availability of a large survey that comprises both the household and institutional population and includes a large set of conditions, including several mental disorders lacking in most previous studies.

## Methods

### Data

Table 1 presents the proportional mortality and disability for each disease group.

Deaths and population by age and gender for the year 2008 for France from Eurostat were derived from the Eurohex website.<sup>7</sup>

**Table 1** Mortality and disability by disease group, age group and sex, France, 2008 based on CépiDc-INSERM-database<sup>8</sup> and Disability Health Survey<sup>9</sup>

	Deaths by cause, % of total						Disability by cause, % of total					
	Men			Women			Men			Women		
	50–64	65–84	85+	50–64	65–84	85+	50–64	65–84	85+	50–64	65–84	85+
Heart disease	12.4	16.5	23.6	7.4	8.7	11.1	9.9	14.8	15.4	4.9	8.9	9.6
CVA	2.8	5.3	7.0	3.3	3.7	5.0	1.6	3.9	4.1	1.0	1.9	2.3
PVD	0.3	0.6	0.8	0.1	0.1	0.2	2.5	3.4	2.7	1.0	1.1	1.3
Other CVD	2.1	2.9	3.7	1.6	1.7	2.1	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.
Musculoskeletal	0.3	0.5	0.7	0.6	0.6	0.8	28.3	21.1	15.5	38.9	33.5	19.9
Cancer	50.3	44.9	25.9	57.6	57.0	51.8	3.1	5.4	2.7	5.2	4.5	2.6
Alzheimer/Parkinson	0.4	3.3	8.3	0.7	1.3	2.7	1.0	2.3	7.3	1.0	3.0	7.5
Other neurological	2.0	1.6	0.9	3.0	2.9	2.6	2.4	1.3	2.2	2.1	1.6	0.9
CNSLD	1.3	2.3	3.0	0.9	1.1	1.4	6.4	6.1	6.3	4.4	3.7	2.4
Acute respiratory infec.	0.9	1.7	4.0	0.7	0.8	1.1	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.
Anxiety and depression	0.2	0.1	0.3	0.3	0.3	0.3	6.5	3.6	3.2	10.8	8.2	4.7
Other mental diseases	3.1	0.8	0.3	1.8	1.5	0.9	1.7	0.9	0.8	0.7	0.4	0.7
Diabetes mellitus	1.6	2.5	2.3	1.6	2.0	2.6	3.1	3.2	1.9	4.0	4.8	2.8
Accidents	12.7	11.8	14.0	12.1	11.7	11.8	6.3	3.8	1.9	2.1	1.9	1.4
Other/background	9.5	5.2	5.3	8.4	6.6	5.3	27.4	29.9	36.1	23.9	26.3	43.7
Total	100	100	100	100	100	100	100	100	100	100	100	100

Note: CVA = cerebrovascular accident (cerebrovascular disease according to ICD-10 terminology). PVD = peripheral vascular disease. Other CVD = other cardiovascular diseases. CNSLD = chronic non-specific lung disease. Alzheimer's/Parkinson groups include dementia. Anxiety and depression are mood disorders. n.a. = not distinguished as separate cause of disability.

Mortality and disability data by cause are presented in table 1. Mortality data by underlying cause of death were obtained from the CépiDc-INSERM-database.<sup>8</sup>

Data on disability by cause were derived in a previous paper<sup>9</sup> based on the French Disability Health Survey ('Enquête Handicap et Santé') 2008–09 which consists of a survey among persons living in private households, HSM and a survey among persons living in in people living in nursing homes, homes for the elderly and mental institutions. Further details of the Disability Health Survey 2008–09 can be found elsewhere.<sup>10,11</sup> Disability prevalence by cause was derived using the attribution method based on the additive hazard model.<sup>1,12</sup> This method takes into account multi-morbidity and that people without reported diseases can still report disability. Further details on the method to estimate disability by cause are given in the Supplementary material.

### Definition of disability

To define disability we used the Global Activity Limitation Indicator (GALI), which is used to calculate HLY across Europe for health monitoring and target setting.<sup>5,13</sup> The GALI is a single question 'For at least the last 6 months, have you been limited because of a health problem in activities people usually do?' aiming to capture long-term limitation (>6 months), and with three severity levels: none, limited but not severely and severely limited. The reliability and validity of the GALI have already been reported.<sup>14–19</sup> People were considered to be disabled (unhealthy) when they reported any limitation. This is the cut-off used in the HLY measure in Europe.

### Causes of disability

We included the following groups of chronic conditions as causes of disability: heart diseases, cerebrovascular accident, peripheral vascular disease (PVD), cancer, chronic non-specific lung diseases (CNSLD), musculoskeletal diseases, Alzheimer's and Parkinson's disease, other neurological diseases (multiple sclerosis, and other unspecific neurological problems), depression and anxiety, other mental diseases (autism, schizophrenia and other unspecified psychiatric impairments), diabetes mellitus and accidents. These causes were selected considering the availability of diseases in the survey and main

disability causes in prior research.<sup>6,20,21</sup> Supplementary table S1 presents the disease groups and diseases within each group.

### Cause of death

We grouped causes of death similarly to the standard groupings for causes of death, however because not all diseases that are disabling are fatal (and vice-versa) we made some adjustments. For completeness we included causes of death that do not cause long-term disability or were not distinguished in the disability survey, such as acute respiratory infections and other circulatory diseases. Causes of disability that are not common causes of death, such as musculoskeletal disorders, are included in the cause of death classification, but with virtually absent mortality.

### Life table and decomposition methods

Life expectancy with GALI disability, i.e. ULYs at age 50 for men and women were estimated by the Sullivan method. This method uses the gender-specific prevalence of disability in each age group to divide the number of person-years years in the standard life table into years with and without disability.<sup>16,22</sup>

The contribution of specific conditions to gender difference in ULYs was estimated by a life table decomposition tool which partitions the difference in ULYs into additive contributions of causes.<sup>1</sup> The decomposition analysis assessed the difference in ULY because of smaller (higher) total mortality rates and/or disability prevalence (by age) from a given cause, in women relative to men. First, the difference in the number of unhealthy person-years (by age) was decomposed into two parts: the first part reflecting the smaller (larger) number of person-years ('mortality effect') and the second part reflecting the smaller (higher) prevalence of disability ('disability effect'). Second, the mortality and disability effects were decomposed by age. For mortality, the decomposition method distinguishes between the age of origin (i.e. the age where mortality differences originate) and the age at destination (i.e. age groups to which person-years are added or removed). For decomposition by age, the age at origin is used,<sup>1</sup> which gives the same result as the decomposition method of Andreev et al.<sup>23</sup> Third, each age effect was decomposed by cause (disease contribution). The

decomposition tool in R, including the decomposition by age, is provided in the Supplementary material. The decomposition analyses also output the decomposition of HLY, which is presented as Supplementary material (Supplementary table S2) for comparison.

## Results

Life expectancy of women aged 50 was 36.3 (95% CI 36.3–36.4) years and of men 30.4 years (95% CI 30.4–30.4) (table 2). Life expectancy with GALI disability was 19.0 years (95% CI: 18.4–19.6) for women compared to 14.2 years (95% CI 13.6–14.8) for men. The gender gap in total life expectancy was 5.9 years (95% CI 5.9–6.0) of which 4.8 (95% CI 4.0–5.7) were ULY.

Table 2 also shows the contribution of mortality differences ('mortality effect') and disability differences ('disability effect') to the gender difference in ULY. The lower mortality and higher disability effect increased ULY in women relative to men. The contribution of the mortality effect was 4.0 years and the disability effect was 0.8 year.

**Table 2** Gender difference in ULYs at age 50, and the contribution of mortality and disability differences for men and women, France, 2008

	ULY Years, (95% CI)
Men	14.2 (13.6 to 14.8)
Women	19.0 (18.4 to 19.6)
Difference	
Women–men	4.8 (4.0 to 5.7)
Contribution of mortality and disability differences	
Mortality effect	4.0 (3.9 to 4.2)
Disability effect	0.8 (–0.1 to 1.7)

**Table 3** Decomposition of gender differences (women–men) in life expectancy with disability (ULY) at age 50, into mortality and disability effects and total effect by cause, France 2008

	Mortality effect (95% CI)	Disability effect (95% CI)	Total effect (95% CI)
Cardiovascular diseases			
Heart disease	0.76 (0.75 to 0.77)	–0.82 (–1.37 to –0.27)	–0.06 (–0.60 to 0.49)
CVA	0.13 (0.13 to 0.14)	–0.23 (–0.45 to 0.00)	–0.10 (–0.31 to 0.13)
PVD	0.03 (0.03 to 0.03)	–0.26 (–0.48 to –0.07)	–0.23 (–0.44 to –0.03)
Other cardiovascular diseases	0.10 (0.10 to 0.10)	n.a	0.10 (0.10 to 0.10)
Musculoskeletal diseases	0.00 (0.00 to 0.00)	1.84 (0.96 to 2.94)	1.84 (0.96 to 2.94)
Cancer	1.75 (1.74 to 1.77)	0.09 (–0.23 to 0.43)	1.84 (1.54 to 2.18)
Neurological diseases			
Alzheimer/Parkinson	0.04 (0.04 to 0.04)	0.08 (–0.17 to 0.28)	0.12 (–0.14 to 0.32)
Other neurological diseases	0.05 (0.04 to 0.05)	–0.04 (–0.22 to 0.12)	0.00 (–0.17 to 0.17)
Respiratory diseases			
CNSLD	0.14 (0.14 to 0.14)	–0.41 (–0.84 to 0.03)	–0.27 (–0.70 to 0.17)
Acute respiratory infections	0.10 (0.09 to 0.10)	n.a	0.10 (0.09 to 0.10)
Mental diseases			
Anxiety and depression	0.00 (0.00 to 0.00)	0.63 (0.20 to 1.04)	0.63 (0.20 to 1.04)
Other mental diseases	0.06 (0.06 to 0.07)	–0.07 (–0.22 to 0.03)	–0.01 (–0.01 to –0.02)
Diabetes mellitus	0.08 (0.07 to 0.08)	0.22 (–0.15 to 0.59)	0.30 (–0.07 to 0.66)
Accidents	0.28 (0.27 to 0.28)	–0.32 (–0.63 to –0.02)	–0.05 (–0.35 to 0.26)
Other/background	0.50 (0.48 to 0.51)	0.08 (–0.27 to 0.33)	0.58 (0.22 to 0.83)
Total	4.0 (3.9 to 4.2)	0.8 (–0.1 to 1.7)	4.8 (4.0 to 5.7)

Note: CVA = cerebrovascular accident (corresponding to cerebrovascular diseases according to the ICD-10 terminology). PVD = periphery vascular diseases. CNSLD = chronic non-specific lung disease. n.a = not available, no separate cause of long-term disability. Gender differences in ULY can originate from higher disability and/or from lower mortality from the condition (extending the time at risk of disability from any cause). The cause-specific mortality effects indicate the origin of the female excess in overall ULY resulting from lower female mortality, although these diseases are not the causes of the ULY themselves. The total effect refers to the disease-specific origin of the gender differences in ULY, either by higher (lower) disability from this condition, of by lower mortality from this condition, extending the time at risk of disability from any conditions.

Further decomposition of the mortality effect and disability effect of ULY by cause is presented in table 3. Of the total mortality effect of 4.0 ULYs, 1.8 ULYs were due to women's lower mortality from cancer, 0.8 ULY from heart disease and 0.3 ULY from accidents. Table 3 also shows gender differences in the disability effects of different diseases. Higher disability from musculoskeletal diseases and anxiety-depression for women than men increased the gender gap in ULY by 1.8 and 0.6 ULY, respectively. The gender gap in ULY was reduced due to lower disability in women from: heart disease by 0.8 ULY, CNSLD by 0.4 ULY and accidents by 0.3 ULY. The sum of the mortality and disability effects showed that musculoskeletal diseases (due to their higher contribution of disability in women) and cancer (due to their lower contribution of mortality in women) had the greatest contribution to the longer ULY in women.

## Discussion

### Summary of findings

In our study, French women spend 4.8 more years with disability than men, mainly because women live longer and are exposed to disability in these additional years. This survival advantage in women reflects their lower mortality from cancer and cardiovascular diseases, and to a smaller extent accidents and respiratory diseases, which together explained nearly 75% of women's excess ULY. In addition, a small disability effect, reflecting higher prevalence of disability in women, contributed to <1 excess ULY in women. This disability effect results from the opposing contributions of specific conditions: musculoskeletal diseases and anxiety-depression contributed more to disability in women than men; and cardiovascular diseases, CNSLD and accidents contributed more to disability in men.

### Strengths and weaknesses

The strength of our study includes considering disparities in both disability and mortality and using a very comprehensive and large

survey on disability and disease in the entire population. This study used disability data by cause derived from a survey of people living in private households and institutions and included a wide range of conditions including the consequences of injuries and mental diseases. We also had access to very detailed cause-of-death data, which allowed us to make cause of death groups that best matched with the diseases groups in the survey.

Some limitations of our study must be considered. First, data on disability and diseases were self-reported. However, for most chronic diseases, self-report is fairly accurate,<sup>24,25</sup> with lowest accuracy for arthritis.<sup>24,26</sup> Moreover, there is no consistent evidence that men and women form assessments of their health in different ways.<sup>27</sup> Higher disability prevalence in women was also found in several performance-based measures.<sup>28,29</sup> Second, in contrast to the causes of death, which are obtained from the death certificate, the causes of disability in our study were based on a statistical attribution of disability to diseases based on cross-sectional survey data. While this attribution took into account competing causes of disability and the presence of disability in people without any diseases, we cannot ascertain that all diseases were present before the onset of disability. In particular for depression, it cannot be ruled out that disability is the cause and not the result of depression.<sup>30</sup> Third, our data did not allow us to use a cohort perspective nor to study changes over time. The Sullivan method uses a period perspective which involves the stationary assumptions,<sup>31</sup> i.e. constant age-specific hazard rates and constant births equal to the radix of the life table. Simulation studies have shown that these assumptions have a minor influence on the results, unless large changes have occurred in mortality and/or disability in the study period.<sup>31–34</sup> Therefore, the Sullivan method is routinely used to calculate life expectancy with(out) disability. For the decomposition method, based on the Sullivan method, it is noteworthy that it identifies the extent to which differences in disability prevalence and total mortality (in each age group) contribute to ULY differences, but not the contribution of differences in incidence of disability, recovery from disability and survival.<sup>1</sup>

### *Interpretation and comparison with prior studies*

The decomposition analyses assessed which conditions contributed to excess ULY in women. The focus was the origin of these differences and we took into account the excess ULY that can originate from higher disability and/or from lower mortality from the condition (extending the time at risk to disability from any cause). The latter does not provide insight into the conditions that contribute to the ULY resulting from the lower female mortality.

Women experience lower mortality, even after the advent of ill-health,<sup>35</sup> and explanations for this include biological (hormonal and genetic), behavioral and social differences.<sup>25,29,36</sup> It is plausible that the same factors explain both lower mortality and lower disability from cardiovascular diseases (e.g. hormonal, genetic, smoking, alcohol consumption and diet), respiratory diseases (e.g. smoking) and accidents (e.g. risk taking and alcohol consumption), supported by the finding that the lower contribution of these conditions to disability in women was due to lower disease prevalence since the disabling impacts of these conditions did not differ by gender.<sup>9</sup> Our study found opposite contributions for musculoskeletal diseases, diabetes and mental diseases. Compared to men, higher disability in women from these conditions contributed to more ULY in women. For musculoskeletal diseases and diabetes this reflected both higher disease prevalence and higher disabling impact in women than in men,<sup>9</sup> perhaps due to higher frequency of obesity and lower levels of physical activity in French women.<sup>37</sup> Differences in body composition (bone mass, muscle strength) and hormonal differences may also have contributed to a higher susceptibility of women to musculoskeletal diseases.<sup>38</sup> The higher contribution of mental diseases to disability in women than men mainly reflected differences in disease prevalence.<sup>9</sup> These gender differences in the

contribution of mental disease are widely acknowledged but less understood.<sup>39</sup>

Similar to our study, prior decomposition analyses of gender differences in life expectancy with disability<sup>1–3,6</sup> showed a larger gender gap in ULY than in HLY due to the combination of lower mortality and higher disability, both extending ULY in women as compared to men. Two previous studies looked at the contributions of diseases to gender differences in life expectancy with disability.<sup>1,6</sup> Our study confirmed that arthritis contributed most to the longer life expectancy with disability in women than in men because of the disability effect while cancer was the main contributor to longer ULY in women because of the mortality effect. However, the earlier Dutch study<sup>1</sup> showed no contribution from heart diseases to the disability effect, while the Belgium study<sup>6</sup> confirmed our findings of a lower contribution of heart diseases to disability in women for the year 2008, and additionally showed that this effect changed over time from a higher contribution in 2001 for women than for men, to a lower contribution in more recent periods. The differences with the Dutch findings may also reflect differences in timing of the study and the study population. The Dutch study<sup>1</sup> used data from the early 1990s, and since then survival from heart disease has increased substantially, persons who previously died from the disease are now more likely to survive. In addition, the Dutch study<sup>1</sup> excluded the institutionalized population which may explain the absence of the gender difference in the contribution of heart disease.<sup>9</sup>

Prior research on the male–female health-survival paradox has pointed to the role of different diseases: men are more likely to die from fatal diseases and women suffer more from non-fatal diseases.<sup>4,25,27,40</sup> Our study confirmed that higher disability from non-fatal diseases, such as musculoskeletal diseases and anxiety-depression in women contributed to more ULYs in women, but that this effect was partly nullified by higher prevalence of disability in men from the traditional life-threatening diseases, such as cardiovascular diseases, respiratory diseases and accidents. A second explanation for the health-survival paradox given in the literature is that women live longer and for that reason face more disability.<sup>4</sup> Our study confirmed that lower mortality tended to increase ULY in women relative to men, and added that this mortality effect was larger than the disability effect.

### *Conclusions, implications and recommendations*

We find that both elements of the health-survival paradox, higher disability and lower mortality, contribute to the excess ULYs in French women compared to men, although women's mortality advantage was the main contributor. However, within the small net contribution of the disability effect, diseases such as musculoskeletal diseases or anxiety-depression have a substantial positive contribution, though this is offset by the negative contribution of heart diseases, respiratory diseases and accidents. Diseases that are traditionally seen as male fatal diseases also cause disability in men and tend to reduce the gender gap in ULY.

Our study shows that diseases have a different impact on gender differences in ULY, depending on whether the disease is mainly life-threatening (such as cancer), mainly disabling (such as musculoskeletal) or both (such as heart diseases). A life-threatening disease reduces ULY as people live shorter lives and therefore are less exposed to disability from any cause. A disabling disease increases ULY as persons experience disability from this disease. Whether a disease which is both life-threatening and disabling reduces or increases ULY depends on the relative size of both effects.

An implication of our findings for the strategy to reduce the gender gap in ULY is that the largest reduction in the female disadvantage in ULY could be obtained by reducing male mortality disadvantage. However, this would not reduce ULY in women and would increase ULY in men in the total population. Instead, there is a strong need to target prevention of diseases that cause disability,



including musculoskeletal diseases which contribute most to disability in both men and women. This includes policies and interventions that reduce obesity by targeting sedentary behavior, physical inactivity and unhealthy diets. Moreover, reducing the disabling consequences of diseases is increasingly important, as people live longer with diseases that cause disability. Better disease management, more use of assistive devices and technological developments and adaptation of the environment are examples to reduce the burden of disability in people with diseases. This may not only reduce the gender gap in life expectancy with disability, but at the same time avoid future increases in disability for both genders.

## Supplementary data

Supplementary data are available at *EURPUB* online.

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*Conflicts of interest:* None declared.

## Key points

- French women's disadvantage in ULYs compared to men is predominantly due to women's lower mortality from cancer, cardiovascular and respiratory diseases and accidents.
- Women's higher disability contributes moderately to their excess ULYs.
- The substantial disabling effect of musculoskeletal diseases in women's ULY is offset by the lower contribution of heart and respiratory diseases, and accidents compared to men.

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## Does health literacy explain the link between structural stratifiers and adolescent health?

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**Background:** The concept of *health literacy* (HL) may help us to better understand the mechanisms leading to health disparities, and to focus on the factors that can be influenced. However, not much is yet known about how HL is related to health disparities, or whether the association exists among adolescents. The aim of the study was to examine the associations between structural stratifiers, HL and health indicators among adolescents. **Methods:** The nationally representative Finnish *Health Behaviour in School-aged Children* survey data ( $n=3833$ ) were collected from 13- and 15-year-old pupils in the spring of 2014. Pearson correlation coefficients were calculated, and separate path models were estimated for the perceived health and health behaviour measures. **Results:** HL was found to be an independent factor explaining disparities in health. Higher HL was related to positive health outcomes. HL also acted as a mediator between health behaviours and structural stratifiers, except for gender. School achievement and educational aspirations were among the factors explaining the HL level, which in turn, explained the health indicators. **Conclusions:** The concept of HL is of use in understanding health disparities. In particular, HL provides a mechanism via which school achievement and educational plans affect health outcomes. Low HL places adolescents who have poor school achievement and who do not intend to continue on an academic path in an unequal position with respect to their health.

## Introduction

Adolescent health, and disparities in health status, track forward to adulthood.<sup>1,2</sup> Although adolescence is seen as a critical period in many global health agendas, the health of adolescents has received less attention than that of other age groups.<sup>3</sup> In research on disparities, the concept of *health literacy* (HL) as a set of competencies (e.g. critical thinking, self-awareness and citizenship) to promote and sustain health may help in understanding the disparities better,<sup>4</sup> and in deciding the factors that can be influenced. As HL is something that can be developed and learned, it may be a crucial factor in addressing avoidable and unfair health disparities.<sup>5</sup> However, not much is known about precisely how HL is related to health disparities,<sup>4,6,7</sup> or whether the association already exists among adolescents.

Health disparities are caused by many factors, some of which consist of *structural stratifiers*, with their proxy indicators such as income, access to education, social class, gender, ethnicity and employment opportunities.<sup>2,8</sup> According to Galobardes et al.<sup>9</sup> the stratifiers determine ‘which resources and goods are distributed to and accumulated over time by different social groups’ (p. 21), and inequality in the distribution causes health differences.

The latest report of the international Health Behaviour in School-aged Children (HBSC) study<sup>1</sup> confirms that many of the structural stratifiers examined among adults are relevant to health disparities among young people, but they have not received that much attention. According to the report, poorer health outcomes are more prevalent among older children and among children from less affluent families. As the children become older, they are less physically active, they drink and smoke more, and perceive their health as worse than that of younger children. Children from less affluent families consistently perceive their health as poorer. They brush their teeth less often, and eat fewer vegetables and fruits.<sup>1</sup> The health differences explained by inequalities in socioeconomic status have increased during the 21st century.<sup>10</sup> However, gender comparisons show that while girls report lower health and life-satisfaction, more health complaint, and less physical activity, they brush their teeth more often, drink less alcohol, smoke less and eat more fruit and vegetables than boys.<sup>1</sup> These findings confirm the argument of Braveman et al.<sup>11</sup> that ‘although health disparities are systematic, a socially disadvantaged group will not necessarily fare worse on all health indicators, and might fare better on some’ (p. s152).