The Nexus between Pollution and Obesity and the Magnifying Role of Media Consumption: International Evidence from GMM Systems Estimates

Cristiana Tudor

Abstract: The aim of this paper is to uncover the associations between air pollution, media consumption, and the prevalence of obesity. Based on data availability, this study draws on an unbalanced panel of 28 countries and develops and extracts relationships through robust System-General Method of Moments (Sys-GMM) estimators that account for the dynamic nature and high persistence of the variables of interest. In light of previous findings, economic development, trade openness, and government consumption are included as controls in the dynamic panel models. The estimation results consistently indicate that pollution is a strong determinant of obesity, a link that remains robust through the alternative proxies for pollution (i.e., total greenhouse gas emissions (GHG) and carbon (CO₂) intensity of energy generation). However, CO₂ intensity shows the strongest association with obesity. Furthermore, the findings indicate that media consumption is an independent and significant driver of obesity, whilst its inclusion among regressors further magnifies the impact and significance of the pollution factor. Moreover, the combined effect of media consumption and pollution significantly contributes to spurring obesity in all model specifications. Thus, a vicious cycle emerges between air pollution, media consumption, and obesity, with synergistic detrimental health effects. The current findings highlight the importance of continuing and consistent efforts to mitigate pollution and reach related low-carbon policy targets. Moreover, for the sustainable reduction and prevention of obesity, these efforts should be complemented by policy interventions and public campaigns aimed at “healthy” media consumption, such as encouraging regular physical exercise and healthy nutrition.

Keywords: obesity; pollution; media consumption; interaction; economic development; system-GMM

1. Introduction

Air pollution is now regarded as the world’s most serious environmental health concern, responsible for 7 million deaths worldwide each year ([1,2]). Moreover, recent evidence highlights that chronic exposure to air pollution can impact every organ in the human body, complicating and increasing pre-existing health disorders [3]. As such, pollution is acknowledged as a significant driver for mortality and disease [4]. Furthermore, statistics disturbingly show that more than 99% of the global population lives in areas with air pollution levels that surpass the World Health Organization limits, as of 2019 [5,6].

Concurrently, obesity remains a serious public health concern [7], often regarded as a worldwide epidemic [8–11], given estimations that indicate 25% of the world’s population is overweight, among which a third suffers from obesity [12]. The rate of early mortality due to a high body mass index (BMI) increased between 1990 and 2015 from 41.9 to 53.7 per 100,000 individuals, whereas a high BMI caused four million deaths globally over the same period, among which 60% of individuals had obesity and, in the majority of cases, died as a result of cardiovascular diseases [13]. The positive link between an increased risk of
death from cardiovascular disease and an unhealthy body weight has been repeatedly documented [14–16]. This relationship also emerges from our study sample, with Figure 1 reflecting a clear positive association between the prevalence of obesity and mortality at the world level.

![Figure 1.](image)

**Figure 1.** Obesity (percentage of population with a body mass index (BMI) > 30% in all adult population) and mortality, most recent year of available data per country. The red line represents the fitted linear regression line, while the dark grey area displays the 95% confidence interval for predictions from a linear model. Source of data: World Bank’s Development Indicators (WDI) database.

Additionally, the link between diabetes and weight gain has also been established [17]. Moreover, empirical evidence also confirms that there is a significant association between obesity and increased risk for certain cancers [18]. Furthermore, an additional non-trivial issue is that obesity and associated comorbidities may have a significant impact on healthcare expenditures [19], with estimates indicating relative economic burdens ranging from 0.09% to 0.61% of gross domestic product (GDP) at the country level [20].

Considering the above causal relationships, it is critical to identify causes of unhealthy body weight and develop strategies for long-term reduction and prevention.

Specifically, air pollution can cause an unhealthy body weight by causing metabolic dysfunction, the emergence of chronic disease, and the disruption of regular physical exercise [21]. However, the literature investigating the transmission channels between the two paramount factors impacting world population health remains surprisingly limited. In this narrow literature, [22] showed that traffic-related air pollution was positively and significantly connected with an increase in body mass index and obesity in children. [23] confirmed the association between air pollutants, obesity, and diabetes. More recently, [24] also documented that early-life exposure to air pollution is positively linked to an increase in the risk of developing overweight and obesity in childhood. In turn, [25] offered a potential transmission channel by arguing that air pollution is positively linked to sedentary behaviors. This is further reinforced by [26], who agreed that behavioral channels can explain the link between pollution and obesity, as air pollution may either decrease physical activity or may prevent people from engaging in physical activity. Additionally, [27]
revealed another mechanism, as they concluded that thyroid function parameters, which in turn can cause obesity, are linked to exposure to climate and air pollution.

Our study contributes to extending this area of research by exploring the relationship between alternative pollution factors and obesity for a global panel of countries, and by employing a robust mix of control variables. A preliminary assessment of the link between pollution and obesity based on our data sample indicates a positive relationship between the two main variables of interest (Figure 2).

Figure 2. CO$_2$ intensity (kg per kg of oil equivalent energy use) and obesity (percentage of population with a BMI > 30% in all adult population) for most recent year of available data per country. The red line represents the fitted linear regression line, while the dark grey area displays the 95% confidence interval for predictions from a linear model. Source of data: World Bank’s Development Indicators (WDI) database.

Moreover, we build on previous studies that conclude that increased media consumption is a driver for a sedentary lifestyle ([28,29]). In particular, there are well-established links between using the Internet for leisure and engaging in sedentary behavior in both children and adults ([30–33]). In this context, it can be reasonably assumed that increased media consumption, through the promotion of sedentary habits, raises the risk of obesity. We further link this hypothesis with the documented positive association between pollution and unhealthy body weight. We thus hypothesize that the combined effect of media consumption and pollution on obesity through behavioral channels such as physical inactivity can be synergistic. Consequently, another novelty of the current research relies upon the introduction of an interaction factor including a pollution proxy and a media consumption proxy, thus investigating whether increased media consumption and concurrent pollution exposure magnify this health problem for the sample of countries in this study.

Furthermore, in deciding on the control group, we consider previous evidence that links lower socio-economic status (SES) to increased exposure to air pollution [2], as well as previous findings that document positive associations between lower SES, high media consumption, and prevalence of overweight and obesity ([34–36]). Additionally, government consumption and globalization (trade openness) are expected to covary with
the focal independent variables in light of previous results (among others, [37–40]), and are thus used as control or confounding variables [41].

This paper adds to the extant literature in several ways. First, it explores the relationship between pollution and obesity based on a global (albeit narrow, due to data unavailability) panel of countries by employing alternative factors to proxy for pollution and introducing a relevant mix of explanatory variables that has not been used before. Second, it proposes an interaction factor to assess a link that has not been investigated thus far, although indications point toward its relevancy, i.e., the combined effect of pollution and media consumption on obesity. To achieve its goals, this study employs the Generalized Method of Moment (GMM) for alternative model architecture estimation. Thus far, the GMM is acknowledged as the strongest technique for investigating dynamic interactions between variables [42]. Of note, the estimator carries the additional advantage of eliminating the need for distributional assumptions and resolving significant issues with panel data estimations [43].

The results provide updated evidence to highlight that obesity is persistent and is highly impacted by pollution (additionally, the relationship remains robust against alternative proxies). Furthermore, the findings indicate that media consumption is an independent significant driver for obesity, its combined effect with pollution contributes to significantly increasing obesity, and its inclusion as an independent factor further magnifies the effect of pollution. These findings have important policy implications, indicating that positive externalities emerge from global efforts to decarbonize economies, as well as pinpointing the importance of healthy media consumption, for whose attainment policy interventions and education campaigns are needed. The interplay between the two strategies can achieve sustainable low-carbon development and sustainable obesity reduction and prevention.

2. Material and Method

2.1. Data and Variables

The data for all variables in this study were collected from the World Development Indicators (WDI) database of the World Bank.

Pollution was the main explanatory variable in our analysis of obesity occurrence in a country, which was in turn represented by the prevalence of adult obesity, i.e., the percentage of adults ages 18 and over with a body mass index (BMI) of 30 kg/m² or higher (WDI code: SH.STA.OB18.MA.ZS). This research used two alternative indicators to proxy the environmental pollution of a country. First, we employed one of the most commonly encountered proxies for pollution, i.e., total greenhouse gas emissions (GHG) ([44,45]). Second, we used the intensity of pollutant emissions, an indicator that is often used to compare the environmental impact of various fuels or activities. The ratio of carbon dioxide per unit of energy, or the amount of carbon dioxide emitted as a result of using one unit of energy in production, is known as carbon intensity, and it is often employed as a proxy for environmental (under)performance [46].

Furthermore, data for the following explanatory factors were also extracted: the percentage of Internet users in the total population (as a proxy for media consumption), GDP per capita as a proxy for the average socio-economic characteristics of the population, government final consumption expenditure, and trade openness.

Table 1 centralizes the variables employed in the empirical investigations, showing abbreviations, variables’ codes in the WDI database, and description.
Table 1. Variable description.

| Variable Abbreviation | Abbreviation | Variable Code (World Bank WDI Database) | Variable Description |
|-----------------------|--------------|----------------------------------------|----------------------|
| OBES                  | HF.STA.Ob18.ZS | Prevalence of obesity, BMI ≥ 30 (% of population 18+) indicates the percentage of population aged 18 and older with a body mass index (BMI) above 30 |
| GHG                   | EN.ATM.GHGT.KT.CE | Total greenhouse gas emissions (kt of CO₂ equivalent) |
| CO₂ intensity         | EN.ATM.CO2.EG.ZS | CO₂ intensity (kg per kg of oil equivalent energy use) is the ratio of carbon dioxide emitted per unit of energy, or the amount of carbon dioxide emitted as a result of using one unit of energy in production. |
| Media                 | IT.NET.USER.ZS | Individuals using the Internet (% of population). As per the World Bank’s definition, individuals who have utilized the Internet (from any location) in the last three months are included in the indicator. The Internet can be accessed via a computer, mobile phone, personal digital assistant, gaming machine, digital television, and other devices. |
| GDP                   | NY.GDP.PCAP.KD | GDP per capita (constant 2015 USD) |
| Gov Cons              | NE.CON.GOV.TZ | General government final consumption expenditure (% of GDP) |
| TradeOpen             | NE. TRD.GNFS.ZS | Trade openness is the sum of exports and imports of goods and services measured as a share of gross domestic product (% of GDP) |

The final data sample in this research was provided due to the availability of data and includes all countries with available data for the variables of interest for at least three years, which allows for the estimation of dynamic panel models. Consequently, we constructed an unbalanced panel spanning 16 years (i.e., 2000–2015) for 28 countries (see Table 2 for final countries list).

Table 2. The sample.

| Income Level *  | Countries |
|-----------------|-----------|
| High-income     | Austria, Belgium, Croatia, Czech Republic, Denmark, Estonia, Finland, France, Greece, Hungary, Ireland, Italy, Latvia, Netherlands, Norway, Poland, Portugal, Slovak Republic, Slovenia, Spain, Sweden, United Kingdom |
| Upper middle income | Bulgaria, Georgia, Iran, Russian Federation, Turkey |
| Low income      | Nepal     |

* Countries are classified by World Bank income levels.

2.2. Exploratory Data Analysis

One of the most relevant exploratory instruments is histograms of variables, which are presented in Figure 3. The distribution of the obesity variable is closest to normality, indicating that its mean value does not deviate much from its median. Interestingly, the distribution of CO₂ intensity is similar to the distribution of government consumption, suggesting an association between public spending and environmental underperformance.

Further, descriptive statistics for the full sample are centralized in Table 3. The countries in the study sample register a mean level of 16.11 percent for the prevalence of obesity in the adult population, with a high range between a minimum level of 1.70 percent and a maximum of 34.20 percent. The exposure to media, as proxied by the percentage of Internet users, shows the highest variation within the sample, ranging from a minimum level of 0.38 percent to a maximum of 96.81 percent. The economic development, as proxied by the GDP per capita, also presents a high range, spanning from a minimum of USD 585.41 to a maximum of USD 74,355.52. The two variables might contain similar information that will emerge in estimations.
Figure 3. Histograms for the variables of interest.

Table 3. Descriptive statistics.

| Variable     | Mean   | Standard Deviation | Min     | Max          |
|--------------|--------|--------------------|---------|--------------|
| OBES         | 16.11  | 5.79               | 1.70    | 34.20        |
| GHG          | 457,431.83 | 763,866.04        | 11,170.00 | 2,566,170.00 |
| CO2 intensity| 2.21   | 0.62               | 0.29    | 3.49         |
| Media        | 48.20  | 27.82              | 0.38    | 96.81        |
| GDP          | 24,163.88 | 17,353.17         | 585.41  | 74,355.52    |
| GovCons      | 18.97  | 4.06               | 7.90    | 26.08        |
| TradeOpen    | 88.38  | 40.22              | 37.92   | 215.16       |

Source: Estimation results.

Additionally, we noticed a high heterogeneity of obesity prevalence across countries, whereas its time trend is rather smooth through the analysis period, suggesting that whereas time effects are not present, country effects should be considered for robust estimations (Figure 4).

Finally, the correlogram of all variables is presented in Table 4, indicating on one hand that regressors in the model are free from severe collinearity, and additionally that there is a positive association between obesity and pollution. Of note, although some degree of collinearity among predictors is intrinsic, I argue that variables do not suffer from severe multicollinearity (i.e., correlation > 0.8) [47] that would affect estimation results.
Additionally, we noticed a high heterogeneity of obesity prevalence across countries, whereas its time trend is rather smooth through the analysis period, suggesting that whereas time effects are not present, country effects should be considered for robust estimations (Figure 4).

Figure 4. Mean prevalence of obesity (%) by country, including the confidence intervals (Panel A). The evolution of mean prevalence of obesity from 2000 to 2015, with confidence intervals (Panel B). Source of data: World Bank’s Development Indicators (WDI) database.

Table 4. The correlogram of the variables.

|        | GDP  | CO₂ Intensity | GHG  | OBES | TradeOpen | GovCons | Media |
|--------|------|---------------|------|------|-----------|---------|-------|
| GDP    | 1.00 |               |      |      |           |         |       |
| CO₂ Intensity | −0.18 | 1.00          |      |      |           |         |       |
| GHG    | −0.37 | 0.14          | 1.00 |      |           |         |       |
| OBES   | −0.26 | 0.27          | 0.38 | 1.00 |           |         |       |
| TradeOpen | 0.21   | 0.05          | −0.43| 0.10 | 1.00      |         |       |
| GovCons | 0.61  | −0.16         | −0.18| 0.03 | 0.21      | 1.00    |       |
| Media  | 0.56  | −0.24         | −0.29| 0.26 | 0.41      | 0.64    | 1.00  |

2.3. Method

This section describes the general form of the model(s), presents relevant details on dynamic panel data estimation, and establishes the main hypotheses to be tested.

Firstly, to smoothen the data and generate more consistent findings ([47]), all variables were converted to their natural logarithm (ln) form. Consequently, the main relationship of interest based on the previous discussion is given by Equation (1), where all variables enter in their natural logarithm form:

\[
OB_{it} = \gamma_{OBES_i,t-1} + \beta_1 POLLUT_{it} + \beta X'_{it} + \epsilon_{it}
\]  

(1)

where \(OBES_{it}\) is the current level for the obesity indicator in country \(i\), \(POLLUT\) is the time-varying pollution measure in country \(i\) (proxied alternatively by total GHG emissions in the main models and also by CO₂ intensity in further robustness checks), and \(X\) is a vector of control variables including the GDP per capita, trade openness, and government consumption. Lastly, \(\epsilon\) denotes the error terms, the subscript \(i\) \((i = 1, \ldots, n)\) denotes the country \(i\) in the data sample, \(N = 28\), and \(t\) \((t = 1, \ldots, T)\) indicates the time period, such that \(T = 16\).
A second relationship of interest investigates the potential magnifying effect that concomitant exposure to media (proxied by the percentage of Internet users) might have on the obesity prevalence among the population, such that:

\[ OBES_{it} = \gamma OBES_{i,t-1} + \beta_1 LnPOLLUT_{it} + \beta_2 (POLLUT_{it} \ast Media_{it}) + \beta X'_{it} + \varepsilon_{it} \tag{2} \]

The interaction factor that we propose in this study constitutes a novelty, making for a relevant contribution to the existing literature. It relies on previous findings that link pollution and obesity, and, on the other hand, media consumption and obesity through the same transmission mechanism based on behavioral traits, such as a sedentary lifestyle.

The following hypotheses are tested via robust dynamic panel models: \( \beta_1 \) in Equation (1) and \( \beta_1 \) and \( \beta_2 \) in Equation (2) are significant and positive. The impact of control variables, especially the link between income and obesity, is also of interest, serving for comparative purposes to previous findings. Based on empirical results, we expect a negative relationship between the level of economic development and obesity, although this link could depend on the sample of countries included in the investigation.

The presence of the lagged dependent variable on the right side of the equation in both Equations (1) and (2) characterizes a dynamic panel model [48], which raises various econometric issues in estimation that must be properly addressed for reliable inference ([42,49]).

Consequently, the System Generalized Method of Moments (Sys-GMM) estimator proposed by [50,51] is employed in this study to conduct the empirical investigation of the primary impact factors for obesity in the sample of 28 countries. The advantages of the Sys-GMM estimator are non-trivial. First, its alternatives, i.e., OLS and the Within estimators, are known to derive biased and inconsistent estimates in a dynamic context ([52,53]). The bias of these two approaches is eliminated by the system GMM estimator. Moreover, the Sys-GMM estimator also overcomes the issues of fixed effects and endogeneity of regressors, in addition to solving the problem of autocorrelation within individuals ([43,54]). Consequently, Sys-GMM not only provides consistent estimates [55], but it also brings significant efficiency gains in dynamic panel estimations ([56,57]). Not in the least, in comparison to the Difference GMM estimator developed by [58], the Sys-GMM allows for more instruments through an additional assumption under which the first differences of instruments are uncorrelated with fixed effects, thus emerging as a more efficient estimator ([49]). Consequently, the system GMM estimator combines two sets of equations and thus adds the level form moment conditions to the difference form moment conditions, hence reducing biases of the difference-GMM estimators [59].

The following dynamic panel equation estimates model (1) through a dynamic system-GMM analysis:

\[ LnOBES_{it} = \gamma Ln(OBES)_{i,t-1} + \beta_1 LnPOLLUT_{it} + \beta_2 LnGDP_{it} + \beta_3 LnGCons_{it} + \beta_4 LnTradeOpen_{it} + \mu_i + \phi_t + \varepsilon_{it} \tag{3} \]

\( i = 1, \ldots, 28 \) and \( t = 2000, \ldots, 2015 \), where the dependent variable representing obesity is explained by its own lagged value, two alternative proxies for pollution, the interaction factor between pollution and media consumption and other relevant controls, including the GDP per capita, trade openness, and government consumption, while \( \mu_i \) stands for fixed country specific effects, \( \phi_t \) represents time effects and \( \varepsilon_{it} \) is an error term with zero mean.

Similarly, the model specified in Equation (2) takes the following extended form in the Sys-GMM estimation, where different mixes of regressors are alternatively tested:

\[ LnOBES_{it} = \gamma Ln(OBES)_{i,t-1} + \beta_1 LnPOLLUT_{it} + \beta_2 LnPOLLUT_{it} \ast LnMedia_{it} + \beta_3 LnGDP_{it} + \beta_4 LnGCons_{it} + \beta_5 LnTradeOpen_{it} + \mu_i + \phi_t + \varepsilon_{it} \tag{4} \]

It must also be mentioned that the asymptotic efficiency benefits brought about by the Sys-GMM estimator’s added orthogonality assumptions produce a proliferation of instruments that leads to a finite sample bias. As such, as per [60] and similar to the approach of [61], we used only two lags for both the difference and system GMM estimators, and consequently specified a collapsed instrument matrix and robust coefficient covariance.
matrices proposed by [62] (i.e., in the estimation of the “pgmm” function within R’s “plm” package). Within the same function, the “Sys-GMM” estimator is obtained using transformation = “ld” for level and difference. All relevant details for estimating GMM models in R are found in the “plm” package description, which was developed by [63].

Finally, as GMM estimators are consistent if there is no second-order serial correlation for the idiosyncratic errors of the first-differenced equation, we tested for any remaining serial correlation by using the Arellano–Bond (AR) test. Furthermore, to ensure that the validity of instruments assumption is valid, overidentification was also tested by Sargan (1958)’s test of over-identifying restrictions [64], further developed by [65].

Figure 5 provides an overview of the method and the specific steps taken to implement it. R software was employed for the overall method implementation, including all estimations.

Figure 5. Overview of the implemented method. AR1 and AR2 represent the Arellano–Bond tests for first- and second-order autocorrelation in the idiosyncratic errors of the first-differenced equation.

3. Results

Of note, the two-step GMM estimators can be seriously biased downwards in finite samples ([51,66]). In this respect, only one-step System GMM estimates are reported in this section (similar to [49,67,68]), although two-step estimations were also performed. Additionally, we report results from seven alternative model specifications (i.e., M(1) to M(7)) out of many specifications that were estimated, underlining that the main relationships of interest remain significant throughout the specifications. Additionally, as mentioned earlier, the robustness of the System GMM estimators depends both on the assumption that the error term does not have a serial correlation problem and on the validity of instruments. Consequently, these assumptions are verified through the Arellano–Bond test for no serial correlation in the error terms and the Hansen/Sargan J test for the validity of the instruments, which are reported in the bottom rows of all results tables. Moreover, the results tables also include the Wald test of slope coefficients being zero jointly ([69]). The row for the J-test reports the p-values for the null hypothesis of the validity of the overidentifying restrictions, whereas the p-values reported for AR1 and AR2 are the p-values for first- and second-order autocorrelation in the first-differenced residuals equation. In all cases, diagnostic tests confirm that all models are properly specified, as the J test never rejects the
null hypothesis of instrument validity, the Wald test always rejects the null hypothesis that slope coefficients are simultaneously equal to zero, and the AR2 test consistently confirms that there is no second-order auto-correlation in the differenced residuals.

3.1. The Relationship between Pollution and Obesity

Table 5 reports the baseline results of the estimation of Equation (3) with the System GMM estimator over the period of 2000–2015 for the unbalanced panel of 28 countries, where GDP per capita is a control variable in both models, and the GHG emissions are the first proxy for pollution (M(1)), while subsequently the CO₂ intensity is introduced for the same task in M(2).

Table 5. One-step system GMM estimates.

| Dependent Variable: Obesity | M(1) | M(2) |
|----------------------------|------|------|
| Obesity (−1)               | 0.33 *** (0.03) | 0.44 * (0.24) |
| GDP                        | 0.08 * (0.01)   | 0.10 (0.11) |
| GHG                        | 0.09 *** (0.01) |       |
| CO₂ intensity              |     | 0.92 *** (0.33) |
| Hansen/Sargan J-test (p-value) | 0.39 | 0.80  |
| AR1 test (p-value)         | 0.29 | 0.09  |
| AR2 test (p-value)         | 0.28 | 0.32  |
| Wald test for coefficients (p-value) | 0.00 | 0.00  |

Standard errors are reported in parentheses; * significant at 10%; *** significant at 1%. AR1 and AR2 represent the Arellano–Bond tests for first- and second-order autocorrelation in the idiosyncratic errors of the first-differenced equation. Instruments are collapsed; robust inference is performed in the summary.

An analysis of the persistence of the obesity indicator for the countries in the sample confirms the a priori assumption that is based on previous empirical evidence, namely that higher obesity in the previous period contributes to obesity in the current period. The autocorrelation parameter is statistically significant and of higher magnitude in the second model specification (i.e., M(2)) when the CO₂ intensity is used as a proxy for pollution, such that a 1% increase in the lagged obesity factor increases obesity in the current period by about 0.44%.

Moreover, GDP per capita has a positive and significant effect on obesity in M(1) when GHG emissions are employed as a proxy for pollution, but loose statistical significance when CO₂ fulfills this task in M(2).

Most importantly, the coefficients of the pollution factor are positive and very significant for both model specifications, with the magnitude being higher for CO₂ intensity (point estimate of 0.916) than for GHG emissions (point estimate of 0.088). Thus, a 1% increase in carbon dioxide intensity brings an increase in obesity of about 0.92%.

3.2. The Influence of the Interaction Factor

Table 6 reports the results of further System GMM estimates for Equation (4), where GHG in M(3) and CO₂ intensity in M(4) are pollution proxies, GDP per capita is again a regressor in both models, and the interaction factor is introduced as the main impact factor.

The main coefficients of interest remain those corresponding to the pollution factor (GHG/CO₂ intensity) and the interaction factor of the pollution and media consumption variables. As hypothesized, these coefficients are positive and very significant for both model specifications. This implies that pollution is an explanatory factor for obesity and additionally, media consumption is a promoter of obesity for the sample of countries in this study. Specifically, the M(3) model specification includes GHG as a pollution proxy, which is included both as an independent factor and in combination with media consumption (i.e., Internet use) within the interaction factor. The results indicate that the interaction factor increases the impact of the GHG factor from 0.088 to 0.131, whereas the interaction factor itself has a statistically significant impact with a point estimate for its coefficient of
0.03. Similarly, M(4) employs CO$_2$ intensity both by itself and within the interaction factor. The interaction factor has a higher coefficient in M(4), equal to 0.06, whereas the addition of the factor contributes to substantially magnifying the impact of the pollution factor from 0.916 to 1.444. As such, a 1% increase in CO$_2$ intensity brings an increase in obesity of about 1.44% when the interaction between pollution and media consumption is also included.

Table 6. Estimation results: Alternative pollution factor and the addition of the interaction actor.

| Panel A. Dependent Variable: Obesity | M(3) | Estimate |
|-------------------------------------|------|----------|
| Independent Variables               |      |          |
| Obes($\cdot$ 1)                    | 0.20 | ** (0.05) |
| GDP                                | 0.00 | (0.02)   |
| GHG                                | 0.13 | ** (0.02) |
| GHG * Media                        | 0.03 | ** (0.00) |
| Hansen/Sargan J-test (p-value)     | 0.87 |          |
| AR1 test (p-value)                 | 0.27 |          |
| AR2 test (p-value)                 | 0.31 |          |
| Wald test for coefficients (p-value) | 0.00 |         |

| Panel B. Dependent Variable: Obesity | M(4) | Estimate |
|-------------------------------------|------|----------|
| Independent variables               |      |          |
| Obes($\cdot$ 1)                    | 0.54 | ** (0.07) |
| GDP                                | 0.09 | (0.05)   |
| CO$_2$ Intensity                   | 1.45 | ** (0.33) |
| CO$_2$ Intensity * Media           | 0.06 | ** (0.02) |
| Hansen/Sargan J-test (p-value)     | 0.92 |          |
| AR1 test (p-value)                 | 0.37 |          |
| AR2 test (p-value)                 | 0.30 |          |
| Wald test for coefficients (p-value) | 0.00 |         |

** significant at 5%; *** significant at 1%. Instruments are collapsed; robust inference is performed in the summary.

In conclusion, adding the interaction term changed the values of $\beta_1$. The effect of pollution on obesity is now 0.131 + 0.03 * Media consumption when GHG is the pollution proxy. For CO$_2$ intensity, the effect on obesity is estimated as 1.44 + 0.06 * Media consumption. When individuals do not use the Internet, i.e., Media= 0, the effect of pollution is limited at 0.13 (GHG) and 1.44 (CO$_2$ intensity). In other words, assuming no media consumption, the effect of a 1% pollution increase raises the prevalence of obesity by 0.131% and 1.44%, respectively. When all of the adult population in a country uses the Internet, however, the effect of a 1% increase in GHG emissions on obesity is 0.161 (i.e., 0.131 + 0.03 * 1), whereas the effect of a 1% increase in the CO$_2$ intensity is magnified at 1.5 (i.e., 1.44 + 0.06 * 1). Compared to the slope coefficients in M(1) – M(2), the slopes of pollution are significantly higher, and their significance is also increased by the addition of the interaction factor. Consequently, because of the interaction factor, the slope coefficients between pollution and obesity are on one hand higher and on the other hand different for the different media consumption levels.

Additionally, both specifications, M(3) and M(4), show a significant autoregressive coefficient that is, however, impacted differently by the addition of the interaction factor. Thus, when GHG proxies for pollution in M(3), the inclusion of the interaction actor decreases the magnitude of the autoregressive coefficient to 0.20, whereas in M(4) the point estimate for the auto-regressive coefficient is increased beyond the original boundaries to 0.536.
3.3. Further Robustness Checks

Table 7 reports the results of the System GMM estimates for alternative regressor mixes in Equation (4). The main results are still robust to the inclusion of extra variables. Alternative estimations include GDP per capita, trade openness, and government consumption as additional controls.

Table 7. Robustness checks: effect of alternative mix of control variables on obesity. Results from one-step system-GMM dynamic panel estimations.

| Dependent Variable: Obesity | M(5) | M(6) | M(7) |
|-----------------------------|------|------|------|
| Independent Variables      | Estimate | Estimate | Estimate |
| Obes(−1)                   | 0.27 *** (0.05) | 0.63 *** (0.11) | 0.19 *** (0.02) |
| GDP                        | 0.02 (0.03) | 0.01 (0.03) |
| GHG                        | 0.14 *** (0.02) | 0.14 *** (0.04) |
| CO₂ intensity              | 0.50 *** (0.07) |
| Media                      | 0.009 * (0.00) |
| Pollution (GHG/CO₂ intensity) * Media | 0.06 *** (0.00) | 0.01 * (0.00) |
| TradeOpen                  | −0.09 (0.01) |
| GovCons                    | 0.12 * (0.07) |
| Hansen/Sargan J-test (p-value) | 0.70 | 0.91 | 0.83 |
| AR1 test (p-value)         | 0.34 | 0.47 | 0.33 |
| AR2 test (p-value)         | 0.27 | 0.23 | 0.21 |
| Wald test for coefficients (p-value) | 0.00 | 0.00 | 0.00 |

* Indicates significance at 10% level, respectively. *** Indicates significance at 1% level, respectively. Instruments are collapsed; robust inference is performed in the summary.

All specifications show a positive and significant autoregressive coefficient that has the highest magnitude when GDP per capita is not used as a control, implying that economic development does carry a large amount of information related to obesity. As can be seen, the lower bound is equal to 0.19, whereas the upper bound is 0.63.

Most importantly, the Sys-GMM parameters are always positive and significant at 1% for the two measures of pollution; however, the magnitude is higher throughout model specifications when the CO₂ intensity factor is used as a proxy.

The interaction factor is always positive and significant; its significance, as well as its magnitude, is higher when used with the CO₂ intensity proxy for pollution. The use of media (Internet) is by itself a statistically significant factor for obesity at 10%. Moreover, its inclusion in the mix of explanatory variables as an independent factor increases the impact of pollution (i.e., GHG) on obesity from 0.09 to 0.14, whereas its inclusion within the interaction factor also increases the impact of pollution on obesity, reaching 0.13.

Further expanding the control group by means of the addition of the government consumption factor increases the impact of pollution to 0.138, while maintaining its statistical significance at 1%.

The GovCons variable is by itself a significant impact factor for obesity, with a point estimate for its coefficient of 0.117, significant at 10%.
4. Discussion

The findings for the seven Sys-GMM alternative model specifications confirm a priori assumptions, namely that (i) a higher prevalence of obesity in the previous period contributes to a higher prevalence of obesity in the current period, (ii) pollution (all proxies) is the main factor that increases obesity prevalence in the sample of countries in this study, and (iii) media consumption is an independent driver for obesity; moreover, the combined effect of media consumption and pollution on obesity is positive and statistically significant, and also contributes to increasing the magnitude of the pollution impact.

The first line of results agree with previous findings that document the persistent effect of obesity ([70]). Moreover, these findings support the view of the World Obesity Federation as expressed in [71], in that obesity is a chronic, relapsing, progressive disease process and, consequently, they contribute to highlighting that immediate action is needed to prevent and control this worldwide epidemic.

Furthermore, our findings document the positive association between pollution and obesity, with the slope coefficient of the pollution factor remaining robust across different model specifications. In this respect, current research findings are in line with previous studies that conclude that pollution is a driver of obesity (i.e., [22,24]), and thus help to strengthen the evidence that there is a positive association between exposure to pollution and the risk of being overweight ([72,73]). Moreover, current findings resonate on the one hand with [25,26] in that the link between pollution and obesity can be explained by behavioral channels such as physical inactivity or sedentarism and, on the other hand, with [27], in that the link can also be explained by physical channels, such as through the thyroid function parameters. These results further complement the findings of [27], highlighting the importance of public health policy measures to reduce air pollution.

Moreover, we confirm that media consumption (as proxied by the relative number of Internet users) is positively linked to obesity, providing updated evidence to support the findings of [34,74–76], among others. This result is also in line with the conclusions of [28,29,77,78] that indicate that a sedentary lifestyle, concurrent snacking behavior, and reduced sleep duration can act as transmission channels between media consumption and obesity. The current findings can thus be explained by these previously documented links and resonate with the conclusion of [33] that the use of technology for entertainment both reinforces and promotes physical inactivity. We thus additionally argue that the usage of media most often replaces time spent engaging in physical activity, which can also be an explanatory factor for the current results, supported by a recent WHO study ([79]). This potential transmission channel is reinforced by [80] who showed that the amount of time teenagers spend on social media has increased, from 4.4 h per week in 2007 to 11.1 h in 2011.

Additionally, results for most specifications (except for M(1)) show no statistically significant relationship between economic development, as reflected by GDP per capita, and the prevalence of obesity, deviating from previous evidence on the positive association between low socioeconomic status and unhealthy body weight. This discrepancy might be explained, on the one hand, by the data sample used in our analysis. As such, due to the (un)availability of data, our sample is biased toward high-income and upper-middle-income countries. Moreover, this study uses national-level data, as opposed to individual-level data. Furthermore, it has been shown that the positive association between economic development and obesity is mitigated by the education level of the population, being absent or reduced for higher levels of education ([81]). In turn, education is closely linked to economic development ([82]), which in turn, given the sample bias in this study, can explain the current findings. Furthermore, [83] confirmed that GDP is positively related to BMI for levels up to approximately USD 3000 per capita, with no significant relationships beyond these levels. By comparison, our data sample reports a mean value of USD 24,000 per capita, well into the no-relationship zone. Consequently, given that our sample of countries mostly includes rich countries, and in light of previous empirical evidence that positively links education and economic growth ([82,84,85]), we conclude that a possible reason that the current results deviate from previous findings is that they are sample-specific.
On the other hand, the factor corresponding to economic development loses its statistical significance when CO$_2$ intensity is introduced as a proxy for pollution or when the interaction factor is employed. This in turn suggests that CO$_2$ intensity and the number of Internet users contain similar information with the economic development variable, which is plausible and in line with [86], who acknowledged the use of fossil fuels in various activities tied to economic development, which is subsequently driving carbon emissions. The correlation coefficient between media consumption and GDP (i.e., 0.56) also provided a preliminary indication in this respect. As such, the contradiction of previous findings may occur due to the additional variables employed in this study that carry more relevant information for obesity than the income factor.

Finally, the results indicate that the general government final consumption expenditure (% of GDP) is positively associated with obesity. The ratio of government consumption expenditure to gross domestic product is an indicator of government size [87], employed as a production factor in the production function [88]. Its positive impact on obesity in a model that also includes the autoregressive term, pollution (GHG), GDP per capita, and the interaction factor between pollution and media consumption deviates from the results of [89], who encountered a positive relationship between government expenditure and welfare, as represented by increased life expectancy in Pakistan, and from the results of [90], who documented a positive relationship between public expenditure on health care and health status in Lesotho. However, this can on one hand be explained by the significant difference in mean income level between the samples employed in the two studies and the current research. Additionally, this discrepancy could be the result of using different indicators for public consumption, as this study employs aggregate data on government consumption expenditure, whereas the other studies employ public expenditure on health care. The positive contribution of public expenditure to obesity found in this study can reflect indirect effects operating through the impact of government spending on pollution and/or media consumption and their subsequent positive impact on obesity.

5. Conclusions

Over 99% of the world’s population is exposed to air pollution levels that exceed the WHO guidelines, which in turn leads to various health-related negative outcomes. Nonetheless, despite the recognized role of pollution in promoting unhealthy body weight, including obesity, the empirical evidence remains scarce. The aim of this paper is thus to explore the association between pollution and obesity, two paramount indicators for global population health, both responsible for various health problems, premature deaths, and significant economic costs. Drawing on unbalanced panel data for 2000–2015 and employing robust dynamic panel Sys-GMM estimators, this paper also documents trends in the main variables of interest, along with relationships between pollution, obesity, and a relevant group of control variables.

This study documents channels through which pollution impacts obesity, including the roles played by socio-economic development, government consumption, trade openness, media consumption, and the interaction between pollution and media consumption. The evidence suggests that pollution and media consumption are significant drivers for obesity. The effect of pollution is statistically significant and positive across all model specifications, but the positive effect is more robust when CO$_2$ intensity is employed as a proxy for pollution than when the factor representing total GHG emissions plays the same role. Moreover, the introduction of an interaction factor representing the concurrent impact of pollution and media consumption in dynamic panel regressions increases the magnitude of the pollution impact, while also acting as a significant driver for obesity by itself. Other findings indicate that government consumption also contributes to increased obesity, whereas media consumption and CO$_2$ intensity account for a large part of the association between socioeconomic status and obesity.

Two important policy implications emerge from the current results. First, countries should continue their efforts to mitigate pollution levels and meet the increasingly conser-
ervative targets established by international agreements, such as the Paris agreements and the European Green Deal. Policymakers should closely monitor whether countries are on the right track to achieve low-carbon policy targets and also intervene in a timely manner to impose stricter targets. Second, these efforts should be complemented by targeted policies and campaigns aiming to educate the population regarding the importance of physical exercise and healthy nutrition, and the negative consequences of too much and/or unhealthy media consumption. We argue that the synergy between these two approaches would contribute to attaining both sustainable low-carbon development and the sustainable reduction and prevention of obesity.

However, as is the case with most studies, the current findings have to be seen in the light of some limitations. First, due to the unavailability of data on the prevalence of obesity in most world countries, this study draws on a rather narrow and unbalanced panel of countries, which in turn imposes restrictions on statistical procedures and hinders the possibility to generalize the results. Furthermore, although the estimates of the assessed links are robust, the mechanisms are not specifically tested, being offered as potential transmission channels. Second, it should also be acknowledged that the current results emerge from country-level factors and are thus unable to capture potential distinct relationships that might characterize different categories of the population, delineated by socio-economic status or behavioral traits. Consequently, the current findings need to be supplemented by longitudinal human studies aimed at further documenting the effects of air pollution and media consumption on the development of obesity through uncovering specific transmission channels.

Funding: This research received no external funding.

Institutional Review Board Statement: Not applicable.

Informed Consent Statement: Not applicable.

Data Availability Statement: Data employed in this study are publicly available from the World Bank Indicators database.

Conflicts of Interest: The author declares no conflict of interest.

References
1. The United Nations Economic Commission for Europe (UNECE). Air Pollution and Health. 2022. Available online: https://unece.org/air-pollution-and-health#:~:text=Air%20pollution%20is%20now%20considered,pulmonary%20illnesses%20and%20heart%20disease (accessed on 10 March 2022).
2. The European Environment Agency (EEA). Air Pollution: How It Affects Our Health. 2021. Available online: https://www.eea.europa.eu/themes/air/health-impacts-of-air-pollution (accessed on 11 March 2022).
3. Schraunfnelg, D.E.; Balmes, J.R.; Cowl, C.T.; De Matteis, S.; Jung, S.H.; Mortimer, K.; Perez-Padilla, R.; Rice, M.B.; Riojas-Rodriguez, H.; Sood, A.; et al. Air pollution and noncommunicable diseases: A review by the Forum of International Respiratory Societies’ Environmental Committee, Part 2: Air pollution and organ systems. *Chest* 2019, 155, 417–426. [CrossRef]
4. Daiber, A.; Kuntic, M.; Nahad, O.; Delogu, L.G.; Rohrbach, S.; Di Lisa, F.; Schulz, R.; Münzel, T. Effects of air pollution particles (ultrafine and fine particulate matter) on mitochondrial function and oxidative stress—Implications for cardiovascular and neurodegenerative diseases. *Arch. Biochem. Biophys.* 2020, 696, 108662. [CrossRef]
5. World Health Organization (WHO). Ambient (Outdoor) Air Pollution. 2021. Available online: https://www.who.int/en/news-room/fact-sheets/detail/ambient-(outdoor)-air-quality-and-health (accessed on 12 March 2022).
6. World Health Organization (WHO). New WHO Global Air Quality Guidelines Aim to Save Millions of Lives from Air Pollution. 2021. Available online: https://www.who.int/news/item/22-09-2021-new-who-global-air-quality-guidelines-aim-to-save-millions-of-lives-from-air-pollution (accessed on 10 March 2022).
7. Lam, T.M.; Vaartjes, I.; Grobbee, D.E.; Katsenberg, D.; Lakerveld, J. Associations between the built environment and obesity: An umbrella review. *Int. J. Health Geogr.* 2021, 20, 7. [CrossRef]
8. Kohn, M.; Booth, M. The worldwide epidemic of obesity in adolescents. *Adolesc. Med. Clin.* 2003, 14, 1.
9. James, P.T. Obesity: The worldwide epidemic. *Clin. Dermatol.* 2004, 22, 276–280. [CrossRef]
10. Mitchell, S.; Shaw, D. The worldwide epidemic of female obesity. *Best Pract. Res. Clin. Obstet. Gynaecol.* 2015, 29, 289–299. [CrossRef]
11. Di Cesare, M.; Sorić, M.; Bovet, P.; Miranda, J.J.; Bhatta, Z.; Stevens, G.A.; Lacmaña, A.; Kengne, A.-P.; Bentham, J. The epidemiological burden of obesity in childhood: A worldwide epidemic requiring urgent action. BMC Med. 2019, 17, 212. [CrossRef]

12. World Health Organization (WHO). Obesity and Overweight. 2018. Available online: https://www.who.int/news-room/factsheets/detail/obesity-and-overweight (accessed on 10 March 2022).

13. Afshin, A.; Forouzanfar, M.H.; Reitsma, M.B.; Sur, P.; Estep, K.; Lee, A.; Marczak, L.; Mokdad, A.H.; Moradi-Lakeh, M.; Naghavi, M.; et al. Health effects of overweight and obesity in 195 countries over 25 years. N. Engl. J. Med. 2017, 377, 13–27.

14. Balkau, B.; Deanfield, J.E.; Després, J.P.; Bassand, J.P.; Fox, K.A.; Smith, S.C., Jr.; Haffner, S.M. International Day for the Evaluation of Abdominal Obesity (IDEA) a study of waist circumference, cardiovascular disease, and diabetes mellitus in 168,000 primary care patients in 63 countries. Circulation 2007, 116, 1942–1951. [CrossRef]

15. Calle, E.E.; Thun, M.J.; Petrelli, J.M.; Rodriguez, C.; Heath, C.W., Jr. Body-mass index and mortality in a prospective cohort of US adults. N. Engl. J. Med. 1999, 341, 1097–1105. [CrossRef]

16. Oster, G.; Edelson, J.; O’Sullivan, A.K.; Thompson, D. The clinical and economic burden of obesity in a managed care setting. Am. J. Manag. Care 2000, 6, 681–689.

17. Runge, C.F. Economic consequences of the obese. Diabetes 2007, 56, 2668–2672. [CrossRef]

18. Renehan, A.G.; Tyson, M.; Egger, M.; Heller, R.F.; Zwahlen, M. Body-mass index and incidence of cancer: A systematic review and meta-analysis of prospective observational studies. Lancet 2008, 371, 569–578. [CrossRef]

19. Behan, D.F.; Cox, S.H.; Lin, Y.; Pai, J.; Pedersen, H.W.; Yi, M. Obesity and Its Relation to Mortality and Morbidity Costs. Society of Actuaries. 2010. Available online: https://www.soa.org/globalassets/assets/Files/Research/Projects/research-2011-obesity-relation-mortality.pdf (accessed on 4 May 2022).

20. Müller-Riemenschneider, F.; Reinhold, T.; Berghöfer, A.; Willich, S.N. Health-economic burden of obesity in Europe. Eur. J. Epidemiol. 2008, 23, 499–509. [CrossRef]

21. An, R.; Ji, M.; Yan, H.; Guan, C. Impact of ambient air pollution on obesity: A systematic review. Int. J. Obes. 2018, 42, 1112–1126. [CrossRef]

22. Jerrett, M.; McConnell, R.; Wolch, J. Traffic-related air pollution and obesity formation in children: A longitudinal, multilevel analysis. Environ. Health 2014, 13, 49. [CrossRef]

23. Yang, C.; Kong, A.P.S.; Cat, Z.; Chung, A.C. Persistent organic pollutants as risk factors for obesity and diabetes. Curr. Diabetes Rep. 2017, 17, 132. [CrossRef]

24. de Bont, J.; Díaz, Y.; de Castro, M.; Cirach, M.; Basagaña, X.; Nieuwenhuijsen, M.; Duarte-Salles, T.; Vrijheid, M. Ambient air pollution and the development of overweight and obesity in children: A large longitudinal study. Int. J. Obes. 2021, 45, 1124–1132. [CrossRef]

25. Seo, M.Y.; Kim, S.H.; Park, M.J. Air pollution and childhood obesity. Clin. Exp. Pediatr. 2020, 63, 382. [CrossRef]

26. Tainio, M.; Andersen, Z.J.; Nieuwenhuijsen, M.J.; Hu, L.; De Nazelle, A.; An, R.; Garcia, L.M.G.; Goenka, S.; Zapata-Diomedi, B.; Bull, F.; et al. Air pollution, physical activity and health: A mapping review of the evidence. Environ. Int. 2021, 147, 105954. [CrossRef]

27. Zeng, Y.; He, H.; Wang, X.; Zhang, M.; An, Z. Climate and air pollution exposure are associated with thyroid function parameters: A retrospective cross-sectional study. J. Endocrinol. Invest. 2021, 44, 1515–1523. [CrossRef]

28. Coon, K.; Goldberg, J.; Rogers, B.; Tucker, K.L. Relationships between use of television during meals and children’s food consumption patterns. Pediatrics 2001, 107, e7. [CrossRef]

29. Vandewater, E.; Shim, M.; Kaplovitz, A. Linking obesity and activity level with children’s television and video game use. J. Adolesc. 2004, 27, 71–85. [CrossRef]

30. Vandelenotte, C.; Sugiyama, T.; Gardner, P.; Owen, N. Associations of leisure-time internet and computer use with overweight and obesity, physical activity and sedentary behaviors: Cross-sectional study. J. Med. Internet Res. 2009, 11, e1084. [CrossRef]

31. Sisson, S.B.; Broyles, S.T.; Baker, B.L.; Katzmarzyk, P.T. Television, reading, and computer time: Correlates of school-day leisure-time sedentary behavior and relationship with overweight in children in the US. J. Phys. Act. Health 2011, 8, S188–S197. [CrossRef]

32. Serrano-Sanchez, J.A.; Marti-Trujillo, S.; Lera-Navarro, A.; Dorado-Garcia, C.; Gonzalez-Henriquez, J.J.; Sanchis-Moysi, J. Associations between screen time and physical activity among Spanish adolescents. PLoS ONE 2011, 6, e24453.

33. Woessner, M.N.; Tacey, A.; Levinger-Limor, A.; Parker, A.G.; Levinger, P.; Levinger, I. The evolution of technology and physical inactivity: The good, the bad, and the way forward. Front. Public Health 2021, 9, 655491. [CrossRef]

34. Kleiser, C.; Schaffrath Rosario, A.; Mensink, G.; Prinz-Langenohl, R.; Kurth, B.M. Potential determinants of obesity among children and adolescents in Germany: Results from the cross-sectional KiGGS Study. BMC Public Health 2009, 9, 46. [CrossRef]

35. Reidpath, D.D.; Burns, C.; Garrard, J.; Mahoney, M.; Townsend, M. An ecological study of the relationship between social and environmental determinants of obesity. Health Place 2002, 8, 141–145. [CrossRef]

36. Wamala, S.P.; Wolk, A.; Orth-Gomér, K. Determinants of obesity in relation to socioeconomic status among middle-aged Swedish women. Prev. Med. 1997, 26, 734–744. [CrossRef]

37. Halkos, G.E.; Paizanos, E.A. The effect of government expenditure on the environment: An empirical investigation. Ecol. Econ. 2013, 91, 48–56. [CrossRef]
71. Bray, G.A.; Kim, K.K.; Wilding, J.P.H.; World Obesity Federation. Obesity: A chronic relapsing progressive disease process. A position statement of the World Obesity Federation. *Obes. Rev.* 2017, 18, 715–723. [CrossRef]  
72. Bloemsma, L.D.; Wijs, A.H.; Klopman, J.O.; Janssen, N.A.; Smid, H.A.; Koppelman, G.H.; Brunekreef, B.; Lebret, E.; Hoek, G.; Gehring, U. The associations of air pollution, traffic noise and green space with overweight throughout childhood: The PiAMA birth cohort study. *Environ. Res.* 2019, 169, 348–356. [CrossRef] [PubMed]  
73. McConnell, R.; Shen, E.; Gilliland, F.D.; Jerrett, M.; Wolch, J.; Chang, C.C.; Lurmann, F.; Berhane, K. A longitudinal cohort study of body mass index and childhood exposure to secondhand tobacco smoke and air pollution: The Southern California Children’s Health Study. *Environ. Health Perspect.* 2015, 123, 360–366. [CrossRef] [PubMed]  
74. Kline, S. Countering children’s sedentary lifestyles: An evaluative study of a media-risk education approach. *Childhood* 2005, 12, 239–258. [CrossRef]  
75. Taylor, J.P.; Evers, S.; McKenna, M. Determinants of healthy eating in children and youth. *Can. J. Public Health* 2005, 96, S22–S29. [CrossRef]  
76. Murer, S.B.; Saarsalu, S.; Zimmermann, J.; Herter-Aeberli, I. Risk factors for overweight and obesity in Swiss primary school children: Results from a representative national survey. *Eur. J. Nutr.* 2016, 55, 621–629. [CrossRef]  
77. Boyce, T. The media and obesity. *Obes. Rev.* 2007, 8, 201–205. [CrossRef]  
78. Robinson, T.N.; Banda, J.A.; Hale, L.; Lu, A.S.; Fleming-Milici, F.; Calvert, S.L.; Wartella, E. Screen media exposure and obesity in children and adolescents. *Pediatrics* 2017, 140 (Suppl. S2), S97–S101. [CrossRef]  
79. CNBC. WHO Report Warns that Adolescents Get too Little Exercise as Screen Time Replaces Physical Activity in Homes across the World. 2019. Available online: https://www.cnbc.com/2019/11/21/who-warns-adolescents-get-too-little-exercise-as-screen-time-prevails.html (accessed on 22 June 2022).  
80. Shimoga, S.V.; Eryana, E.; Rebello, V. Associations of social media use with physical activity and sleep adequacy among adolescents: Cross-sectional survey. *J. Med. Internet Res.* 2019, 21, e14290. [CrossRef]  
81. Kinge, J.M.; Strand, B.H.; Vollset, S.E.; Skirbekk, V. Educational inequalities in obesity and gross domestic product: Evidence from 70 countries. *J. Epidemiol. Community Health* 2015, 69, 1141–1146. [CrossRef]  
82. Kruss, G.; McGrath, S.; Petersen, I.H.; Gastrow, M. Higher education and economic development: The importance of building technological capabilities. *Int. J. Educ. Dev.* 2015, 43, 22–31. [CrossRef]  
83. Egger, G.; Swinburn, B.; Islam, F.A. Economic growth and obesity: An interesting relationship with world-wide implications. *Econ. Hum. Biol.* 2012, 10, 147–153. [CrossRef] [PubMed]  
84. De Meulemeester, J.L.; Rochat, D. A causality analysis of the link between higher education and economic development. *Econ. Educ. Rev.* 1995, 14, 351–361. [CrossRef]  
85. Pastor, J.M.; Peraita, C.; Serrano, L.; Soler, A. Higher education institutions, economic growth and GDP per capita in European Union countries. *Eur. Plan. Stud.* 2018, 26, 1616–1637. [CrossRef]  
86. Jorgenson, A.K. Economic development and the carbon intensity of human well-being. *Nat. Clim. Chang.* 2014, 4, 186–189. [CrossRef]  
87. Hajamini, M.; Falahi, M.A. The nonlinear impact of government consumption expenditure on economic growth: Evidence from low and low-middle income countries. *Cogent Econ. Financ.* 2014, 2, 948122. [CrossRef]  
88. Barro, R.J. Government spending in a simple model of endogeneous growth. *J. Political Econ.* 1990, 98, S103–S125. [CrossRef]  
89. Chaudhry, I.S.; Moe, M.S.; Sheikh, S.M.; Idrees, S. Asymmetric effect of FDI and public expenditure on population health: New evidence from Pakistan based on non-linear ARDL. *Environ. Sci. Pollut. Res.* 2022, 29, 23871–23886. [CrossRef]  
90. Akinkugbe, O.; Mohanee, M. Public health expenditure as a determinant of health status in Lesotho. *Soc. Work. Public Health* 2009, 24, 131–147. [CrossRef]