GLOBAL STABILITY OF DIABETIC CHILDREN WITH ELECTRONIC GADGETS USING LYAPUNOV FUNCTION

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ABSTRACT: Electronic gadgets such as smart mobile phones, tablets and television are very easily available to all the people including children. Children tend to remain busy for long time with these gadgets causing lack of physical activities resulting obesity. The obesity is significant cause for type II diabetes mellitus, especially in children. We have designed the system of non-linear differential equations to study the effect of electronic gadgets on children younger than 10 years. The endemic equilibrium point is obtained for the proposed model and its global stability is established using Lyapunov function. The numerical simulation is worked out the pros and cons for the use of electronic gadgets at a younger age through data. We study the global stability of the system using Lyapunov function.

Keywords: electronic gadgets; obesity; Diabetes Mellitus II; threshold; global stability.

2010 AMS Subject Classification: 34D20, 92D30.

1. Introduction

Children have easy access of electronic gadgets such as mobile phones, tablets and television. Such gadgets keep the children occupied and quiet for long time. Even parents use these gadgets for children’s food time and to pacify them as parents themselves are unable to spare time with the children. Due to these factors, children are inclined to spend more time, called ‘screen time’, with electronic gadgets. Higher screen time results in lower physical activity time for children. If higher

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fraction of children has more screen time, other children do not get partners for physical activities. As a result, more children have more screen time. Thus, a child having significant screen time is infectious phenomenon.

Study by Zhang, et al. [13] found that TV watching is linked to increased risk of childhood obesity. Increase of 1 hours daily screen time increases obesity risk by 13% in children. Another study by Rosiek [1] indicates that additional hour of watching TV per week results in increase of obesity risk by 3%. Watching TV influences eating habits by way of commercials and multimedia contents, in addition to physical inactivity. Higher screen time results in combination of physical inactivity and unhealthy high calorie food causing increased risk of obesity. A survey by Rosen [4], finds significant association between TV watching hours and body fat mass. Every extra hour/day spent in TV watching results in approximately 1 Kg increase in body fat. This rise can be attributed to not only physical inactivity but eating patterns as well. In the survey by Dennison and Edmunds [2], it is also found that quantity and calories are higher when food is taken while watching TV. Higher TV watching time displaces time available for physical activity, reduces energy expenditure relative to energy intake and leads to obesity. Moreover, according to Jenvey [12], immaturity of young children increases their temptation for foods of poor nutritional quality advertised often during children’s television programs resulting in unhealthy food preferences, and dietary imbalances associated with obesity.

According to WHO, [6], the number of obese children (0 to 5 years) was 32 million globally in 1990, which went up to 41 million in 2016. Developing countries have rate of increase in obese children more than 30% higher than that of developed countries.

As per study conducted by Harvard and published on [9], TV watching is linked to obesity and reduction in TV watching time results in weight control and it is recommended that children and teenagers should not spend more than 2 hours per day in TV/media watching. In addition to children getting obese due to higher screen time, there could be a small fraction of overweight children by birth.

According to WHO, [6], Diabetes mellitus-II (DM-II) and obesity in children have high correlation. Obese children are likely to have Diabetes mellitus-II. Obesity in childhood is linked to a wide range of serious health
issues and an increased risk of premature onset of illnesses, including diabetes and heart disease. Upper socioeconomic strata and urban areas of India is witnessing increase in childhood obesity. As found in WHO survey by Praveen and Tandon [10], there is a link between childhood obesity and the diabetes epidemic in India as indicated by emerging literature. Obese children are at higher risk of having abnormalities that are recognized as signs to diabetes, such as subclinical inflammation, insulin resistance and metabolic disorder.

In addition, there could a very small fraction of children having DM-II due to other reasons such as genetic parameters.


2. **Mathematical model**

A mathematical model for children spending significant time on electronic gadgets becoming diabetic is formulated. The notations, description and their parametric values are described in Table 2.1.

<table>
<thead>
<tr>
<th>Notations</th>
<th>Description</th>
<th>Parametric value (in percentage)</th>
</tr>
</thead>
<tbody>
<tr>
<td>$B$</td>
<td>Recruitment rate of children in system</td>
<td>10</td>
</tr>
<tr>
<td>$\beta_1$</td>
<td>Rate of children getting into spending more time on electronic gadgets</td>
<td>40</td>
</tr>
<tr>
<td>$\beta_2$</td>
<td>Rate of children being obese by birth</td>
<td>3</td>
</tr>
<tr>
<td>$\beta_3$</td>
<td>Rate of children being diabetic by birth</td>
<td>0.1</td>
</tr>
<tr>
<td>$\gamma_1$</td>
<td>Rate of children getting obese due to spending significant time on electronic gadgets</td>
<td>60</td>
</tr>
<tr>
<td>$\gamma_2$</td>
<td>Rate of children not remaining obese though spending significant time on electronic gadgets</td>
<td>40</td>
</tr>
<tr>
<td>$\eta$</td>
<td>Rate of obese children becoming diabetic</td>
<td>70</td>
</tr>
<tr>
<td>$\mu$</td>
<td>Rate at which children leave the system</td>
<td>70</td>
</tr>
</tbody>
</table>
Figure 2.1 depicts dynamics of children in various compartments.

![Diagram of children in compartments](image)

This model gets transformed into set of non-linear differential equations given in (1).

\[
\begin{align*}
\frac{dC}{dt} &= B - \beta_1 CE_g - \beta_2 C - \beta_3 C - \mu C \\
\frac{dE_g}{dt} &= \beta_1 CE_g - \gamma_1 E_g + \gamma_2 O - \mu E_g \\
\frac{dO}{dt} &= \beta_2 C + \gamma_1 E_g - \gamma_2 O - \eta O - \mu O \\
\frac{dD}{dt} &= \beta_3 C + \eta O - \mu D
\end{align*}
\]

Adding the differential equations (1), we have

\[
\frac{d}{dt}(C + E_g + O + D) = B - (C + E_g + O + D) \mu \geq 0
\]

This gives, \( \lim_{n \to \infty} \sup (C + E_g + O + D) \leq \frac{B}{\mu} \). Therefore, feasible region for equations (1) is

\[
\Lambda = \{(C, E_g, O, D) : (C + E_g + O + D) \leq \frac{B}{\mu}, C > 0, E_g, O, D \geq 0\}
\]

We calculate the basic reproduction number using next generation matrix method given by Diekmann et al. [5]. The next generation matrix method is finding spectral radius of matrix \( FV^{-1} \)
where $F$ and $V$ are the Jacobian matrices of $f$ and $v$ evaluated with respect to each compartment at an equilibrium state.

To find the equilibrium state, we solve the equation (1), which gives endemic equilibrium point

$$E^* = \{C^*, E_g^*, O^*, D^*\}$$

where $C^* = \frac{b \pm \sqrt{b^2 - 4ac}}{2a}$ with

$$a = \beta_1[(\beta_2 + \beta_3 + \mu)(\eta + \mu) + \gamma_2(\beta_3 + \mu)]$$
$$b = -[B\beta_1(\eta + \gamma_2 + \mu) + \beta_3(\gamma_1(\eta + \mu) + (\eta + \gamma_2 + \mu)\mu) + \beta_2(\eta(\gamma_1 + \mu) + (\gamma_1 + \gamma_2)\mu)]$$
$$c = B[\eta(\gamma_1 + \mu) + (\gamma_1 + \gamma_2 + \mu)\mu]$$

$$E_g^* = \frac{B - (\beta_2 + \beta_3 + \mu)C^*}{\beta_1C^*}$$
$$D^* = \frac{C^*(\beta_1(\mu + \eta + \gamma_1 + \gamma_2) + \eta(\beta_2 - \gamma_1))\mu + \eta \gamma_1 B}{(\eta(\gamma_1 + \mu) + (\mu + \gamma_1 + \gamma_2)\mu)\mu}$$
$$O^* = \frac{(-\mu \gamma_1 + \mu \beta_2 - \gamma_1 \beta_2)C^* + By_1}{\eta(\gamma_1 + \mu) + (\mu + \gamma_1 + \gamma_2)\mu}$$

Let $X = \{C, E_g, O, D\}$. Then $\frac{dX}{dt} = f(X) - v(X)$ where $f(X)$ denotes the rate of new children in the system and $v(X)$ denotes the rate of transmission of children from one compartment to another compartment which is given by

$$f(X) = \begin{bmatrix} \beta_1 CE_g \\ 0 \\ 0 \\ 0 \end{bmatrix}$$
$$v(X) = \begin{bmatrix} \gamma_1 E_g + \mu E_g - \gamma_2 O \\ -\beta_2 C - \gamma_1 E_g + \gamma_2 O + \eta O + \mu O \\ -\beta_3 C - \eta O + \mu D \\ -B + \beta_1 CE_g + \beta_2 C + \beta_3 C + \mu C \end{bmatrix}$$

Derivatives of $f$ and $v$ give matrices $F$ and $V$ of order $4 \times 4$ defined as $F = \left[ \frac{\partial F_i}{\partial X_j} \right]$ and $V = \left[ \frac{\partial V_i}{\partial X_j} \right]$; for $i, j = 1, 2, 3, 4$
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So, \( F = \begin{bmatrix} \beta_1 C & 0 & 0 & \beta_1 E_g \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix} \) and \( V = \begin{bmatrix} \mu + \gamma_1 & -\gamma_2 & 0 & 0 \\ -\gamma_1 & \mu + \gamma_2 + \eta & 0 & -\beta_2 \\ 0 & -\eta & \mu & -\beta_3 \\ \beta_1 C & 0 & 0 & \beta_1 E_g + \beta_2 + \beta_3 + \mu \end{bmatrix} \),

\[
FV^{-1}(E^*) = \begin{bmatrix} \beta_1 C^2 x_2 (\gamma_2 + \eta + \mu) \\ \beta_1 C^2 \gamma_2 x_2 \\ 0 \\ x \end{bmatrix} \begin{bmatrix} x_1 \\ 0 \\ 0 \\ 0 \end{bmatrix} \begin{bmatrix} \beta_2 \beta_1 C^2 \gamma_2 + (B - C^* x_2) \\ 0 \\ \eta (\gamma_1 + \mu) + (\gamma_1 + \gamma_2 + \mu) \mu \end{bmatrix}
\]

where \( x_1 = \beta_1 \beta_2 \gamma_2 C^2 + B[\eta(\gamma_1 + \mu) + (\gamma_1 + \gamma_2 + \mu) \mu] \) and \( x_2 = (\beta_2 + \beta_3 + \mu) \)

The basic reproduction number \( R_0 \) which is spectral radius of \( FV^{-1} \) and it is given by

\[
R_0 = \frac{\beta_1 C^2 (\gamma_2 + \eta + \mu)(\beta_2 + \beta_3 + \mu)}{\beta_2 \beta_1 \gamma_2 (C^*)^2 + B[\eta(\gamma_1 + \mu) + (\gamma_1 + \gamma_2 + \mu) \mu]}
\]

3. Stability analysis

In this section, local and global stability of the endemic equilibrium point is analyzed.

3.1 Local stability

**Theorem 3.1.** \( E^* \) is asymptotically stable if the following conditions are satisfied:

\[
\gamma_1 + \mu \geq \beta_1 C^*
\]

\[
(\gamma_2 + \eta + \mu)(\gamma_1 + \mu - \beta_1 C^*) \geq \gamma_1 \gamma_2
\]

\[
B \geq C^*(\beta_2 + \beta_3 + \mu)
\]

**Proof.** Jacobian matrix evaluated at point \( E^* \) is

\[
J(E^*) = \begin{bmatrix} -a_{11} & -\beta_1 C^* & 0 & 0 \\ a_{11} - (\beta_2 + \beta_3 + \mu) & -a_{22} & \gamma_2 & 0 \\ \beta_2 & \gamma_1 & -a_{33} & 0 \\ \beta_3 & 0 & \eta & -\mu \end{bmatrix}
\]
where, \( a_{11} = \frac{B}{C^*}, \ a_{22} = -\beta_1 C^* + \gamma_1 + \mu \) and \( a_{33} = \gamma_2 + \eta + \mu \).

The eigenvalues of the above matrix are \( \lambda_1, \lambda_2, \lambda_3 \) and \( \lambda_4 \) where \( \lambda_1 = -\mu \) and \( \lambda_2, \lambda_3 \) and \( \lambda_4 \) satisfies the characteristic polynomial equation \( \lambda^3 + b_1 \lambda^2 + b_2 \lambda + b_3 = 0 \) where

\[
\begin{align*}
b_1 &= a_{33} + a_{22} + a_{11} \\
b_2 &= -\gamma_1 \gamma_2 + a_{33} a_{22} + a_{33} a_{11} - \beta_1 \beta_2 C^* - \beta_1 \beta_3 C^* - \beta_1 \mu C^* + \beta_1 C^* a_{11} + a_{22} a_{11} \\
b_3 &= \beta_1 C^* \gamma_2 \beta_2 - \gamma_1 \gamma_2 a_{11} - a_{33} \beta_1 \beta_2 C^* - a_{33} \beta_1 \beta_3 C^* - a_{33} \beta_1 \mu C^* + a_{33} \beta_1 C^* a_{11} + a_{33} a_{22} a_{11}
\end{align*}
\]

\[
\gamma_1 + \mu \geq \beta_1 C^* \Rightarrow a_{22} \geq 0; \text{ Moreover, } a_{11} \geq 0 \text{ and } a_{33} \geq 0. \text{ Hence, } b_1 \geq 0.
\]

\[
\begin{align*}
b_2 &= -\gamma_1 \gamma_2 + a_{33} a_{22} + a_{33} a_{11} - \beta_1 \beta_2 C^* - \beta_1 \beta_3 C^* - \beta_1 \mu C^* + \beta_1 C^* a_{11} + a_{22} a_{11} \\
&= \gamma_1 + \mu \geq \beta_1 C^*; (\gamma_2 + \eta + \mu) - \beta_1 \gamma_1 \gamma_2 + \beta_1 [B - (\beta_2 C^* + \beta_3 C^* + \mu C^*)] + a_{33} a_{11} + a_{22} a_{11} \\
&\geq 0 \text{ if } \gamma_1 + \mu \geq \beta_1 C^*; (\gamma_2 + \eta + \mu) \geq \gamma_1 \gamma_2 \text{ and } B \geq C^* (\beta_2 + \beta_3 + \mu).
\end{align*}
\]

If \( \gamma_1 + \mu \geq \beta_1 C^*; (\gamma_2 + \eta + \mu) \geq \gamma_1 \gamma_2 \text{ and } B \geq C^* (\beta_2 + \beta_3 + \mu) \) then \( b_1 b_2 > b_1 \) is satisfied.

Hence, by Routh Hurwitz criteria [11], \( \lambda_2, \lambda_3 \) and \( \lambda_4 \) have negative real part and \( \lambda_1 \) is already negative real number and hence \( E^* \) is asymptotically stable.

### 3.2 Global stability

**Theorem 3.2.** (Stability of \( E^* \)) Endemic equilibrium point is globally asymptotically stable.

**Proof.** Consider the Lyapunov function [8]:

\[
L(t) = \frac{1}{2} [(C - C^*) + (E_g - E_g^*) + (O - O^*) + (D - D^*)]^2 \\
= [(C - C^*) + (E_g - E_g^*) + (O - O^*) + (D - D^*)][B - \mu C - \mu E_g - \mu O - \mu D]
\]

Therefore,

\[
L'(t) = [(C - C^*) + (E_g - E_g^*) + (O - O^*) + (D - D^*)][C' + E_g' + O' + D'] \\
= [(C - C^*) + (E_g - E_g^*) + (O - O^*) + (D - D^*)][B - \mu C - \mu E_g - \mu O - \mu D]
\]

Taking \( B = (C^* + E_g^* + O^* + D^*) \mu \)
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\[ L'(t) = -\mu((C-C^*)+(E_g-E_g^*)+(O-O^*)+(D-D^*)) \leq 0 \]

By LaSalle’s Invariance Principle solution [7], \( E^* \) is globally asymptotically stable.

4. Sensitivity analysis

The sensitivity of all parameters is discussed in this section. We use Christoffel formula

\[ \Gamma_{\alpha}^{R_0} = \frac{\partial R_0}{\partial \alpha} \frac{\alpha}{R_0} \]

where \( \Gamma_{\alpha}^{R_0} \) represents change in basic reproduction number (\( R_0 \)) with respect to the parameter \( \alpha \) affecting the model for study of the diseases for normalized sensitivity of the parameters.

Table 4.1 Sensitivity Analysis for threshold \( R_0 \)

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Impact</th>
</tr>
</thead>
<tbody>
<tr>
<td>( \beta_1 )</td>
<td>++</td>
</tr>
<tr>
<td>( \beta_2 )</td>
<td>-</td>
</tr>
<tr>
<td>( \beta_3 )</td>
<td>0</td>
</tr>
<tr>
<td>( \gamma_1 )</td>
<td>-</td>
</tr>
<tr>
<td>( \gamma_2 )</td>
<td>+</td>
</tr>
<tr>
<td>( \eta )</td>
<td>-</td>
</tr>
<tr>
<td>( \mu )</td>
<td>-</td>
</tr>
</tbody>
</table>

N.B. ++ denotes highly sensitive, + stands for sensitive, - stands for negatively related and 0 for no impact.

Rate of children getting into spending more time on electronic gadgets (\( \beta_1 \)) has very high impact on children getting addicted to electronic gadgets and rate of children not remaining obese though spending significant time on electronic gadgets (\( \gamma_2 \)) has positive impact on children getting addicted to electronic gadgets. Children spending significant time to obese rate (\( \gamma_1 \)) has negative impact. Other parameters except for rate of children having diabetes from birth have negative impact. Children born with diabetes do not have visible impact, as this rate is very small.
5. Simulation

In this section, transmission of children between compartments is observed numerically.

Figure 5.1 Duration v/s Number of children in Compartments

Figure 5.1 indicates that there is sharp rise in population of children spending significant time on electronic gadgets followed by rapid fall. Another aspect to observe is that in 1.25 years obese children become diabetic and in 1.81 years children with higher screen time turn obese. Population in obese compartment rises for first 6 months but after that there is rapid fall. Similarly, population of diabetic children increase gradually for 2 years but after that it recedes.

Figure 5.2 Obese to Diabetic children (θ) - duration v/s Number of Diabetic Children

Figure 5.2 shows impact of flow rate $\theta$ (from obese to diabetic) on number of diabetic children. It is observed that this rate has significant impact on number of diabetic children. If children are involved in physical activity then even if they are obese, they may not turn diabetic. This may result in lowering the transmission rate. As indicated by the graph, number of diabetic children remains low with lower transmission rate.
Figure 5.3 Children with significant screen time (E_g) to Obese Duration (γ_1) v/s Number of Obese Children

Figure 5.3 shows the effect of flow rate γ_1 [from compartment E_g (Children with significant screen time) to O (Obese Children)] on number of obese children on time axis. For lower transmission rates, population of obese children is low. It suggests that if children do physical activities along with using electronic gadgets then they may not become obese, which would result in lower transmission rate to obese and in turn lower obese population.

Figure 5.4 Rate of children spending significant screen time (β_1) v/s number of diabetic children

Figure 5.4 suggests that inflow rate of fresh children getting into spending significant screen time has impact on number of diabetic children. However, this is less significant compared to subsequent compartment movement. This indicates that if subsequent movement can be kept low, number of diabetic children would turn out to be low. This can be achieved by supplementing their activities by physical activities and food habits.
6. Conclusion

In this article, impact of children spending significant time of electronic gadgets getting obese and in turn suffer from Diabetes Mellitus – type II is studied. The basic reproduction number has been calculated which helps us to know the phenomenon of children getting obese and in turn becoming diabetic. The system has endemic equilibrium. Local and global stability of the system is studied at that point. Simulation suggest that if movement of children with significant screen time to getting obese and movement from obese to diabetic can be kept low then number of diabetic children turns out to be low. This suggests that children’s activities should be supplemented by physical activities and healthy food habits.

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Conflict of Interests

The authors declare that there is no conflict of interests.

REFERENCES


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