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The recent study published in the journal *Pediatrics* on 20, September 2010 has established the relationship between adenovirus 36 (AD36) which is associated with the common cold and childhood obesity. Gabbert et al (2010) concluded that the higher body weight in children can be associated with the presence of neutralizing antibodies to AD36 (p. 726). The established link between the presence of antibodies and child's obesity gives rise to various questions, including the direction of causality and presence of the link in adults. Establishing the link between childhood obesity and AD 36, the researchers used inductive arguments. They draw the conclusions from specific observations, suggesting various versions of the cause-and-effect relationships between the factors but not ensuring any of them and leaving room for further research of the problem.

Gabbert et al (2010) noted that “ Possibilities for this association include true causality, increased susceptibility to infection within obese children, and predisposition to persistent AD36-specific antibodies after infection” (p. 726).

The premises for drawing the conclusions were the results of a cross-sectional study of a study sample of 50 obese and 50 non-obese children.

The premise was “ The frequency of AD36-specific antibodies was significantly ($P < .02$) greater in obese children (15 [22%] of 67 children) than in nonobese children (4 [7%] of 57 children)” (p.

723). The conclusion from this premise was that the presence of AD-36 antibodies can be associated with obesity in children. Considering the fact that the researchers do not indicate the direction of the causality and do not exclude the reverse link between the overweight and disposition to viral infections, the conclusions can be regarded as logical. Still, the study and its

design have been criticized by a number of researchers. Walsh (2010) noted that “ Weaknesses include the cross-sectional design, which does not allow for conclusions about causality, and the lack of information about the timing of infection” (Adenovirus strain associated with obesity in kids).

Considering the weaknesses of the study design and insufficiency of the study results for defining the causality direction, the inductive argumentation was the best suitable strategy for the study. Along with the association of AD-36 and childhood obesity, there might be some additional factors which complicate the solution of the problem and need to be taken into consideration for developing the appropriate intervention strategies. Scott Kahan, the co-director of the George Washington University Weight Management Program, expresses his opinion concerning the issue, viewing the problem from a new perspective: “ This study is just a snapshot in time, so we can’t say whether having this virus causes people to gain weight or predisposes them to certain behaviors” (Mann, 2010, Virus may be linked to childhood obesity).

Mentioning the behavior characteristics as the intermediate factor having impact on the established relationship can give rise to doubts as to the direct link between the factors. Similar study was conducted in Korea, establishing the same link between AD-36 and childhood obesity (Na et al, 2010, p. 93). Further research is required for establishing the direction of the causality and defining whether the obesity causes immune dysfunction and disposition to infections or the presence of antibodies results in obesity. Though a number of studies have proven the association of AD-36 and childhood obesity, the available data is insufficient for establishing the cause-and-

effect relationships between the two factors and developing the intervention strategies for solving the problem.

Reference List

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