Human adenovirus 36 and weight gain in a rat model

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Obesity is defined as a condition of excessive fat accumulation in adipose tissue that may associated with a higher incidence of chronic diseases, such as heart diseases, hypertension, type 2 diabetes mellitus and several types of cancers, with a significant economic impact on the healthcare system .

The worldwide prevalence of obesity more than doubled between 1980 and 2014. Over 600 million adults were obese in 2014, and it is predicted that 51% of the world's population will be obese by 2030.

Viruses 2015; 7: 3719-40.
Obesity. 2006; 14: 1905-13
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Obesity is a multifactorial disease develops from a complex of interactions among genetic, metabolic, behavioral, as well as environmental factors. Moreover, infection by certain pathogens may be considered as possible causes of obesity .

A variety of recent evidence has been shown a positive relationship between obesity and adenoviruses, with a particular emphasis on the human Ad-36 that has a direct effect on adipose tissue. The present study was conducted to examine the relationship between adenovirus 36 infection and obesity in a rat model.

Table 1. Body weights in the groups

	Infected	Control	P-v
Body weight (g) Baseline	192.8±16.3 (N:48)	195.3±9.0 (N:12)	0.48
At the time of infection	229.0±25.9 (N:48)	232.3±16.6 (N:12)	0.60
12 weeks after infection	304.0±39.0 (N:46)	301.0±36.5 (N:11)	0.82
Change	75.8 ± 27.8	70.8 ± 24.5	0.57

METHODS

Eight-week-old male Wistar rats from the Laboratory Animal Unit of Jundishapur University of Medical Sciences (Ahvaz, Iran) from July 2016 to October 2016 were used for the experiment. They were weighing 170-240 gram (g). Rats were housed in groups of six per cage in a temperature-controlled animal room (20–22 C) on an automatic 12h: 12h light/dark cycle. After one-week acclimatization period, rats were randomly divided into two groups, infected group (48 Rats) and a control group (12 Rats). The rats in the infection group were infected with human Ad-36. All rats were given free access to a normal chow diet and water and were weighed weekly.

RESULTS

At the beginning of the study, the mean and standard deviation (SD) of body weight for infected group was 192.8 ± 16.3 g and in the control group was 195.3 ± 9.0 g (p = 0.48). At the time of infection, the mean body weights were 229.0 ± 25.9 g and 232.3 ± 16.6 g in the infected and the control groups (p = 0.60), respectively.

At 12th week after-infection, while the two study groups had approximately equal food intakes and food disappearance, the mean body weight of the infected group was higher than the control group (304.0±39.0 g vs. 301.0±36.5 g, p = 0.82)

Table 2. Food intake in the groups

	Infected	Control	P-v
Food intake/d (g) Baseline	22.8±1.0	22.6±0.56	0.52
At the time of infection	23.1±1.1	22.8±0.58	0.36
12 weeks after infection	25.9 ± 1.1	25.2±1.6	0.26
Change	75.8 ± 27.8	70.8 ± 24.5	0.57

CONCLUSION

In the present study, infection of rats with Ad-36 had no statistically significant effect on weight gain. Further studies with longer duration of follow-up are suggested. As a model of medical research, rat has many advantages for further investigations about Ad-36-induced obesity. In addition, it is strongly recommended not to rely merely on weight measurement; body composition assessment would be accompanied by weight measurement.